SPECTRUM OF NEUROLOGICAL MANIFESTATIONS IN ACUTE KIDNEY INJURY: A PROSPECTIVE OBSERVATIONAL STUDY IN TERTIARY CARE CENTRE

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Dedicated

to

my family

&

Teachers

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Dr. Arshdeep Kaur Gill

LIST OF ABBREVIATIONS

ACS	**-**	Acute coronary syndrome
AFP	-	Acute flaccid paralysis
ADA	-	Adenosine deaminase
AKI	-	Acute kidney injury
ANCA	-	Anti-neutrophil cytoplasmic antibodies
APACHE	-	Acute physiology and chronic health evaluation
BIA	-	Bioelectrical Impedance Analysis
B/L	-	Bilateral
BUN	-	Blood urea nitrogen
Ca^{++}	-	Calcium
CAD	-	Coronary artery disease
CAM ICU	-	Confusion assessment method for the ICU
CI	-	Confidence interval
CLD	-	Chronic liver disease
CSF	-	Cerebrospinal fluid
CKD	-	Chronic kidney disease
CNS	-	Central nervous system
Cr	-	Creatinine
CT	-	Computed tomography
DA	-	Dopamine
DM	-	Diabetes Mellitus 2
DMCH	-	Dayanand Medical College and Hospital
DNA	-	Deoxyribonucleic acid
DSS	-	Dengue shock syndrome
E.coli	-	Escherichia Coli
eGFR	-	Estimated glomerular filtration rate
EEG	-	Electroencephalography
ESRD	-	End stage renal disease
GBS	-	Guillen barre syndrome
GCS	-	Glasgow coma scale
GFR	-	Glomerular filtration rate
HE	-	Hepatic encephalopathy

HRS	-	Hepatorenal syndrome
HUS	-	Hemolytic uremic syndrome
ICU	-	Intensive care unit
IL	-	Interleukin
KDIGO	-	Kidney disease improving global outcomes
MAP	-	Mean arterial pressure
MMSE	-	Mini-Mental State Examination
MRI	-	Magnetic resonance imaging
MRSA	-	Methicillin resistant staphylococcus aureus
N	-	No
NSAID	-	Non steroidal anti-inflammatory drugs
OR	-	Odds ratio
PRES	-	Posterior reversible encephalopathy syndrome
PTH	-	Parathyroid hormone
qSOFA	-	Quick sepsis related organ failure assessment score
RNA	-	Ribonucleic acid
RRT	-	Renal replacement therapy
SAE	-	Sepsis associated encephalopathy
SBP	-	Systolic Blood pressure
SD	-	Standard deviation
SGA	-	Subjective Global assessment
T3	-	Triiodothyronine
T2W FLAIR	-	T2 weighted fluid attenuation inversion recovery
TNF	-	Tumour necrosis factor
VEGF	-	Vascular endothelial growth factor
WHC	-	Wst haven criteria
Y	-	Yes

ABSTRACT

Introduction: Acute kidney injury is one of the most important causes of morbidity and mortality with a myriad of clinical spectrum including cardiovascular, hematological, neurological and respiratory complications.

Aim of Study: To assess the spectrum of neurological features in Acute kidney injury and correlate it to stages of Acute kidney injury and underlying etiology.

Materials & methods: A prospective observational study was conducted on 200 patients who had Acute kidney injury on presentation. A detailed clinical examination including GCS was documented along with blood counts, chemistry, ABG supplemented by MRI , CT, EEG , NCV and CSF analysis as and when required.

Results: Majority of patients were male (58%) The most common cause of acute kidney injury was infections (59%). 22% % of patients had neurological complications of which the most common was delirium (68%) followed by sleep inversion (52%) and seizures (4%). Hypertension was a risk factor for AKI and its neurological complications. There was no correlation of stages of AKI with presence of neurological features. Presence of sepsis, septic shock, higher qSOFA score, need for inotropes and need for ventilatory support were independent predictors of neurological complications in AKI

Conclusion: Majority of patients with acute kidney injury are males and elderly. Neurological features in patients with acute kidney injury are secondary to AKI and as a part of multiorgan disease process. Hypertension, sepsis, shock, need for ventilatory support and higher q SOFA score are predictors for patients with AKI who are likely to have neurological complications. The spectrum of these neurological features include delirium, sleep inversion, seizures, myoclonus, asterixis, tremors and wasting. The development of neurological complications cannot be predicted by using baseline values of blood urea or creatinine. There is more role of clinical examination and radiological investigations to predict these complications.

Keywords: Acute kidney injury, neurological, creatinine, urine output, sepsis, delirium, seizures, dialysis, stroke, GCS, uremia.

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INTRODUCTION

Acute kidney injury is one of the most common complications associated with remote organ dysfunction in critically ill patients. It is defined as an abrupt (within hours) decrease in kidney function, which encompasses both injury (structural damage) and impairment (loss of function). It is a syndrome that rarely has a sole and distinct pathophysiology. Many patients with acute kidney injury have a mixed aetiology where the presence of sepsis, ischemia and nephrotoxicity often co-exist and complicate recognition and treatment.

Initially wide array of definitions were used to define acute renal failure but a consensus was decided to follow KDIGO criteria which uses the surrogates of kidney function, serum creatinine level and urine output, to define the presence and severity of kidney injury[1]

The latest classification of acute kidney injury proposed by the Acute kidney injury Working Group of KDIGO (Kidney Disease: Improving Global Outcomes), is based on the previous two classifications, and had the aim of unifying the definition of acute kidney injury. By the KDIGO definition, acute kidney injury is diagnosed by an absolute increase in serum creatinine, at least 0.3 mg/dL (26.5 μmol/L) within 48 hours or by a 50% increase in serum creatinine from baseline within 7 days, or a urine volume of less than 0.5 mL/kg/h for at least 6 hour.[1]

EPIDEMOLOGY OF ACUTE KIDNEY INJURY

According to a systematic review (2004-2012) of large cohort studies the pooled incidence rates of acute kidney injury was 21.6% in adults concluding that 1 in 5 adults worldwide experienced acute kidney injury during a hospital admission.[2]

The incidence of Acute kidney injury is 21.6 % in adults with mortality of 23.9 % in adults.[2] It has been found that Acute kidney injury is more

likely to occur in children and healthy adults in developing countries than in developed countries because of a host of socioeconomic and environmental influences.[3] Acute kidney injury can be hospital acquired or community acquired.

Acute kidney injury can range in severity from asymptomatic and transient changes in laboratory parameters of glomerular filtration rate (GFR), to overwhelming and rapidly fatal derangements in effective circulating volume regulation and electrolytes, acid-base composition of the plasma.

The common clinical features are nausea, vomiting, fatigue, oliguria/anuria, fluid overload, metabolic acidosis, hyperkalaemia and altered mental status.

Serum creatinine levels, blood urea and urine output are functional biomarkers that have several limitations. Urine output has a low specificity because this parameter can be influenced by several factors, including hypovolaemia and the use of diuretics. By contrast, serum creatinine level have low sensitivity in previously healthy kidneys, because the serum creatinine levels increase only if at least 50% of the functional nephrons are lost.[4]

Recent evidence is beginning to change the view for acute kidney injury from a single organ failure syndrome to a syndrome where the kidney plays an active role in the evolution of multi-organ dysfunction. Acute kidney injury is not an isolated event, and it results in remote organ dysfunction to the lungs, heart, liver, intestines, and brain. Acute kidney injury leads to distant organ injury through a combination of proinflammatory and oxidative stress-mediated mechanisms. Serum and distal organ cytokine levels (IL1, IL6, IL10 and TNFa) increase in conjunction with Leukocyte trafficking (neutrophil, lymphocyte, and macrophage) and increased oxidative stress (superoxide dismutase,

malondialdehyde, and glutathione depletion. In addition, sodium and water channel dysregulation in the lungs aggravates pulmonary oedema.[5]

Patients with acute kidney injury are at an increased risk of long-term adverse outcomes including mortality, chronic kidney disease, end-stage renal disease, cardiovascular events, and reduced quality of life. Acute kidney injury has been reported to be associated with an increased risk of heart failure, acute myocardial infarction and of stroke.[6]

NEUROLOGICAL MANIFESTATIONS IN ACUTE KIDNEY INJURY

Complex mechanisms of crosstalk between injured kidneys and remote organs such as the nervous system, lungs, liver, heart, gut, brain, and hematologic system have been identified.

Uremic encephalopathy encompasses a wide range of central nervous system abnormalities associated with poor kidney function occurring with either progressive chronic kidney disease or acute kidney injury. The syndrome is likely caused by retention of uremic solutes, alterations in hormonal metabolism, changes in electrolyte and acid-base homeostasis, as well as changes in vascular reactivity, blood–brain barrier transport, and inflammation.

Patients with acute kidney injury frequently develop confusion as one of the earliest neurological disturbances. A florid spectrum of fluctuating clinical signs ranging from headache, visual abnormalities, tremor and asterixis, multifocal myoclonus, chorea, seizures, and coma have been described to occur in setting of acute kidney injury.

However, the cause of these symptoms and signs are multifactorial.

Majority of patients who develop acute kidney injury have underlying Hypertension, Diabetes mellitus 2, CAD and are more likely to be older. Hence there is variable degree of underlying small vessel ischemic brain disease. This makes them more susceptible to cerebral injury in any

setting of loss of cerebral autoregulation e.g., in hypotension/shock or of disturbance of blood brain integrity e.g., in sepsis/liver failure.

Renal replacement has considerably evolved over the years. However, controversies still exist on timing of initiation and selection of patients.

In addition, treatment with renal replacement therapy does not fully ameliorate all the symptoms. Symptoms like sluggishness, memory impairment, and sleep disturbances may persist after Renal replacement therapy.

NEED FOR STUDY

Identifying the neurological features in Acute kidney injury can help stratify patients for management and reduce mortality and morbidity. In low- and middle-income countries, acute kidney injury imposes difficult challenges due to the lack of data on the epidemiology and causes of acute kidney injury, the limited health care to diagnose and treat acute kidney injury, and the poor awareness of the outcomes of acute kidney injury.

- Chronic kidney disease has been widely studied for its neurological spectrum, but Acute kidney injury is uncharted waters in this respect. Isolated studies have been done on neurological features specific to aetiology, but no study has been done in India to analyse the neurological aspect of Acute kidney injury as a single entity. This would be the first study to assess neurological features of Acute kidney injury irrespective of underlying cause and use the data to draw inference about the clinical presentation and correlation if any present with disease outcome.

AIM AND OBJECTIVES

- Determine what percentage of patients with acute kidney injury develop neurological changes.
- Describe The Pattern of Neurological Manifestation in Acute kidney injury.

REVIEW OF LITERATURE

ETIOLOGY OF ACUTE KIDNEY INJURY

The causes of acute kidney injury are generally divided into three major categories:

1) Prerenal acute kidney injury which includes diseases that cause renal hypoperfusion, resulting in decreased function without frank parenchymal damage. It represents the most common form of kidney injury and often leads to intrinsic acute kidney injury if not corrected promptly. Volume loss from Gastrointestinal tract, renal, cutaneous (e.g., burns) and internal or external hemorrhage can result in this syndrome.

2) Intrinsic acute kidney injury which included diseases that directly involve the renal parenchyma. Structural injury in the kidney is the hallmark of intrinsic acute kidney injury, and the most common form is acute tubular injury (ATN) which can be ischemic or cytotoxic.

3) Post renal acute kidney injury which included diseases associated with urinary tract obstruction.

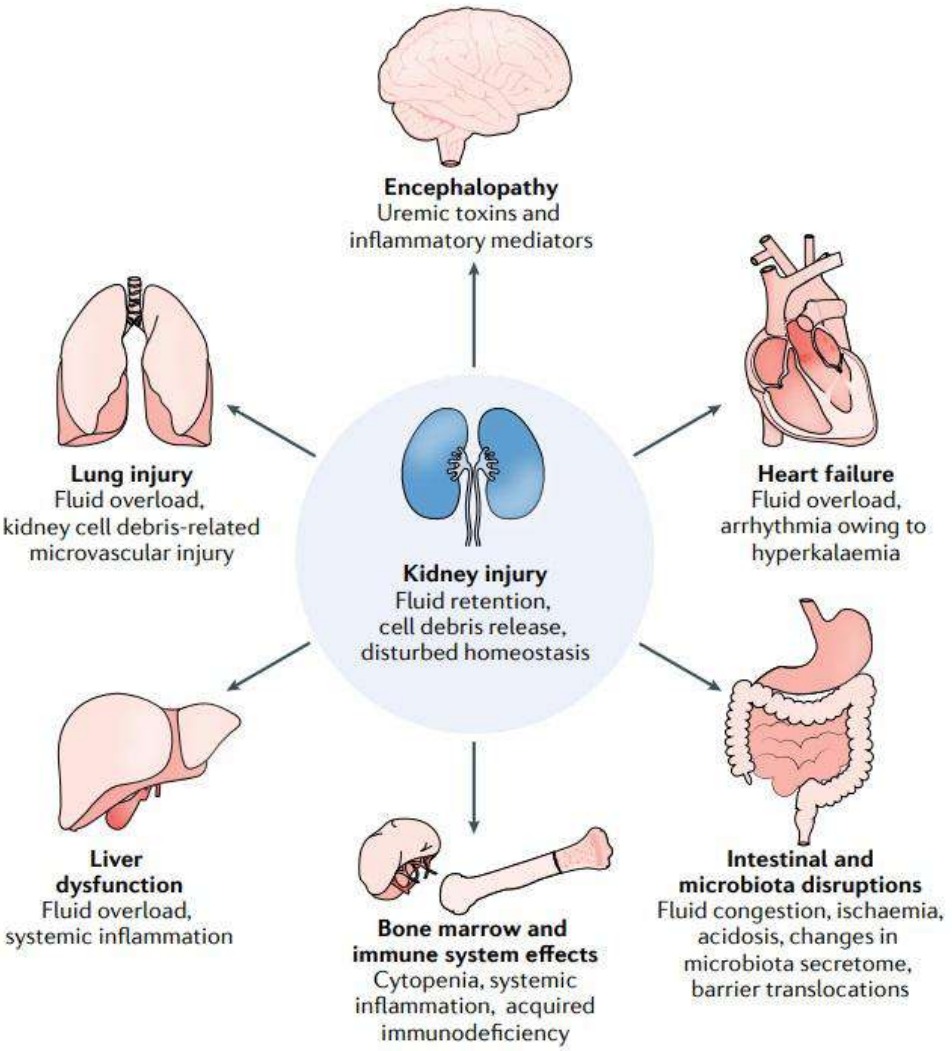
The causes of Acute kidney injury show wide variation in developed and developing countries. Factors contributing to Acute kidney injury such as infectious diseases (that is, tropical febrile illnesses such as diarrhoea, leptospirosis, malaria and dengue fever), envenoming (as a result of snake, spider or insect bites and/or stings), and obstetrical complications are described far more commonly in developing countries than in those from developed countries.[7] In a study done in France on 500 patients with acute kidney injury which included 227 males and 237 females ranging in age from 7 to 81 years the aetiologies or predisposing causes of acute kidney injury were surgical (50%), traumatic (52%), obstetrical (15%) and medical origins (46%).[8] In recent years, new causes have been reported

which include Hanta virus, ingestion of raw fish, quinine hypersensitivity, ecstasy, HIV infection, gelatin infusion and herbal medicine.[9]

CLINICAL PRESENTATION

Patients who develop acute kidney injury can be oliguric or non-oliguric, can have a rapid or slow rise in creatinine levels. This lack of a uniform clinical presentation reflects the variable nature of the injury.

COMPLICATIONS OF ACUTE KIDNEY INJURY


Encephalopathy
Uremic toxins and inflammatory mediators
Lung injury
Fluid overload, kidney cell debris-related microvascular injury
Heart failure
Fluid overload, arrhythmia owing to hyperkalaemia
Kidney injury
Fluid retention, cell debris release, disturbed homeostasis
Liver dysfunction
Fluid overload, systemic inflammation
Bone marrow and immune system effects
Cytopenia, systemic inflammation, acquired immunodeficiency
Intestinal and microbiota disruptions
Fluid congestion, ischaemia, acidosis, changes in microbiota secretome, barrier translocations

SOURCE- Kellum, J. A., Romagnani, P., Ashuntantang, G., Ronco, C., Zarbock, A., & Anders, H. J. (2021). Acute kidney injury. *Nature reviews. Disease primers*, *7*(1), 52. https://doi.org/10.1038/s41572-021-00284-z

1) UREMIA

Accumulation of nitrogen products in body and uremic toxins causes cellular dysfunction and altered mental status.

2) VOLUME OVERLOAD

Patient may present with weight gain and dependent oedema. Resolution of kidney injury maybe associated with increased urine output and volume depletion which is due to osmotic diuresis and incomplete recovery of tubular function.

3) HYPONATREMIA

Iatrogenic administration of hypotonic solutions decreased tubular sodium absorption and decreased water excretion; all contribute to hyponatremia.

4) METABOLIC ACIDOSIS

Retention of organic and other acids and consumption of bicarbonate results in acidosis.

5) HYPERPHOSPHATEMIA

Acute kidney injury is a catabolic state which causes hyperphosphatemia. Destruction of red blood cells is additional source for rise in phosphate levels seen in acute kidney injury.

6) INFECTIONS

An immunocompromised state resulting from renal failure makes the patient more prone for infections.

7) CARDIAC

Cardiac manifestations of acute kidney injury include Pericarditis, Arrhythmias and Pericardial effusion.

In a recently published meta-analysis, Otudayo et al. reported a 58%, 40%, and 15% increased risk of heart failure, myocardial infarction, and stroke, respectively.[10]

The factors that contribute to Acute kidney injury-associated mortality vary with time and differ in the immediate and long-term. Factors that modify the risk of early mortality occurring in-hospital or less than 90 days after Acute kidney injury include the primary diagnosis (such as sepsis), severity of acute illness and the burden of acute nonrenal organ dysfunction.[11]

UREMIC ENCEPHALOPATHY

The term uremic encephalopathy is used to describe the early appearance and dialysis responsiveness of the nonspecific neurologic symptoms of uraemia. The symptoms of uremic encephalopathy may include sluggishness and easy fatigue; daytime drowsiness and insomnia with a tendency toward sleep-inversion; itching; inability to focus or sustain attention or to perform mental tasks; inability to manage ideas and abstractions; slurring of speech; anorexia, nausea, and vomiting; restlessness; imprecise memory; disorientation and confusion with bizarre behaviours.[12] Abnormalities of mental status have been noted to be sensitive indices of acute renal insufficiency, which may progress rapidly into disorientation and confusion if untreated. Fixed attitudes, torpor, and other signs of toxic psychosis are common. Cranial nerve signs such as nystagmus and mild facial asymmetries are usually transient. Asymmetrical variation of deep tendon reflexes is noted along with progression of hyperreflexia, with sustained clonus. Animal models in mice

and canine have attempted to explain the alterations in acute kidney injury and resulting biochemical, vascular, and inflammatory changes in brain.

Classical studies of canine acute uraemia demonstrated significant biochemical alterations in the brain, particularly in calcium and parathyroids concentrations along with regulation of volume status.[13] [14] Adachi induced renal failure by occluding bilateral renal arteries for 60 minutes. 48 hours later brain monoamine turnover was evaluated, and it was found that the turnover of DA in the striatum, mesencephalon and hypothalamus was decreased in these rats. The motor activity of the uraemic rats was significantly impaired.[15]

However the level of azotaemia does not correlate well with neurological impairment.[16] Central nervous system changes including decreased mental status, obtundation and seizures, are one of the classic indications to begin dialysis during Acute kidney injury. Dialysis improves but does not fully correct central nervous system manifestations or other distant organ effects of renal failure, either acutely or chronically.[17] The neurological manifestations in Chronic kidney disease have been well described in studies including stroke, intracerebral microbleeds and cognitive.[18] Few studies have evaluated the neurological features in Acute kidney injury even though the symptoms of encephalopathy progress more rapidly in Acute kidney injury than with chronic kidney disease or ESRD.[19]

PATHOPHYSIOLOGY OF NEUROLOGICAL MANIFESTATIONS

Several mechanisms have been proposed for the neurological manifestations of Acute kidney injury.

1) Impaired blood brain integrity

Acute kidney injury increases production of pro inflammatory cytokines and reduces cytokine clearance hence resulting in pro inflammatory state. [20] [21] Loss of renal function also causes accumulation of toxins which

increases serum osmolality. This promotes VEGF and production of reactive oxygen radicals which cause endothelial damage, upregulation of toll like receptors and disruption of blood brain barrier.[21] [22]

Neurotransmitter derangement

Brain catecholamine concentrations have been found to be depleted in animal models of acute kidney injury.[23] Changes in catecholamine neurotransmitter metabolism have been associated with impaired motor activity.[15]

Endocrine alterations

Free T3 levels tend to fall in acute kidney injury, whereas catecholamines, vasopressin, natriuretic peptides, and the renin–angiotensin– aldosterone axis increase. Renal sympathetic nervous system overactivity has been reported to increase the risk of Hypertension and the development of the posterior reversible encephalopathy syndrome due to ischaemic injury and oedema in the medulla and cerebellum.

Trigger inflammatory cascade.

Systemic inflammatory response in acute kidney injury was recently described by Ratliff et al in 3 waves. The first danger signal is rise in uric acid which is a marker of renal ischemic injury. This triggers secondary response of release of contents of weiber palade bodies including endothelin-1, large multimers of von Willebrand factor, IL-8 and angiopoietin-2.Third wave consists of release of high mobility group box 1 protein and DNA binding protein which causes direct activation of the innate immune system via Toll-like receptors, leading to an intense inflammatory response within hours of ischaemic renal injury.[24]

Acid–base disturbance

Increasing intracellular acidification leads to an increasing affinity of ammonia Km [NH4+] for human glutamate dehydrogenase, causing oxidative deamination of glutamate, and thus altering neurotransmitter balance, with excess ammonia cycling between neurons and astrocytes.[25] Protons can activate acid-sensing ion channels causing an influx of both sodium and calcium into the cell, leading to cell membrane depolarisation and cellular injury and cell death.[26] Low intracellular pH can cause calcium influx resulting in an alteration of neural cell plasticity and increased cell injury. The reduction in pH in the cerebrospinal fluid has additional local vasodilatory effects on the cerebral parenchymal arterioles through activation of large conductant, calcium-sensitive potassium channels.[27]

Organic osmolyte and brain water disturbance

Acute kidney injury results in the retention of the waste products of nitrogen metabolism, seen as urea accumulation. Increased plasma urea leads to increased astrocyte and neuronal urea concentrations. As the intracellular osmolality increases, glial cells and neurons initially compensate by excreting sodium, potassium and calcium and other osmotically active organic anions. In acute kidney injury, however, the time course of increasing serum urea and other azotemic toxins is more rapid , and compensatory defence mechanisms take time to become fully operational.[28] A rat model showed an accumulation of organic osmolytes in the brain tissues of rats within 48 hours of developing acute kidney injury, which was followed by reduction of brain water at 8 hours.[29] This initial response is seen as an increase in volume, with the brain transiently generating osmoles derived from amino acids such as glutamine, taurine and inositol to equalise the osmolality gradient of hyperosmolar state in uraemia.[30] However other studies done as animal models failed to reproduce these findings.[31]

Alteration of drug pharmacokinetics

Many sedatives, opioid analgesics and other drugs with central nervous actions have significant renal clearance and therefore altered metabolism in acute kidney injury. This leads to increased half-lives resulting in potential drug accumulation. Hence the patients with acute kidney injury are at greater risk of developing a confused state.Organic anions and cations are excreted by the kidney by glomerular filtration and tubular secretion. Organic anion transporters (OATs) are substrate specific for endogenous and exogenous anions. Schneider and colleagues reported decreased para-amino Hippurate clearance, a protein bound uraemic toxin, in an animal model of acute kidney injury associated with reduced mRNA expression for OAT1 and OAT3.[32] An increase of G-CSF, and glial fibrillary acidic protein (GFAP) in the cerebral cortex and hippocampus of the brain may serve to recruit neutrophils to sites of neuronal damage.[33]

Renal replacement therapy can help improve encephalopathy due to uraemia by removing azotemic retention products and reducing toxic drug levels. However too rapid a fall in serum urea may paradoxically cause cerebral oedema. Dialysis can directly or indirectly be associated with dialysis dementia, disequilibrium syndrome, aggravation of atherosclerosis, cerebrovascular accidents due to ultrafiltration-related arterial hypotension, hypertensive encephalopathy, Wernicke's encephalopathy, haemorrhagic stroke, subdural hematoma, osmotic myelinolysis, opportunistic infections, intracranial Hypertension and mononeuropathy[17]

While urea is the surrogate for neurotoxins, which are lowered with dialysis, there is no credible evidence linking urea to encephalopathy. Recent studies have shown that indoxyl sulphate can cause vascular inflammation and neurological symptoms.[34]

During a 12-year follow-up of a cohort study Tsai HH et al observed that patients with acute kidney injury exhibited a higher incidence of global cognitive impairment and dementia.[35] Cognitive impairment following acute kidney damage has been found to be a sign of functional or structural changes in the hippocampus.[36]

Lee et al found that a baseline eGFR <60 ml/min/1.73 m^2 was independently related to incidence of stroke.[37] Paul Teschan et al observed that there are quantitative impairments of Central nervous system function that vary directly with the degree of renal failure, improve following onset of maintenance dialysis, and improve still further after successful renal transplantation.[38]

A retrospective study was done by Mos et al where patients with concomitant acute kidney injury and neurological features were selected. The incidence of neurological disturbances and acute kidney injury was 6.6 per 1,000 ICU admissions (95% CI 4.7 - 9.1). Of the 35 patients with neurological disturbances and acute kidney injury, NWA- neurological status attributable to several causes, including acute kidney injury was present in 28 patients. NDA -no other explanation for the neurological status than acute kidney injury was present in 7 patients.[39] It concluded that in critically ill patients with neurological disturbances and acute kidney injury, acute kidney injury is usually only one of the possible factors contributing to the neurological status. The combination of acute kidney injury and neurological disturbances indicated a poor prognosis with Renal replacement having little effect on neurological improvement or mortality.[39]

One of the cardinal manifestations of uraemia is day–night reversal. Kidney failure is characterized by substantial changes in circadian rhythm, sleep architecture and endogenous melatonin release. Evidence suggests a direct association between severity of sleep problems and biochemical measures of kidney failure.[40] CKD is associated with disordered circadian

rhythms of sleep, blood pressure, and proteinuria. In human subjects, worsening renal function is associated with later onset, shorter duration, and increased fragmentation of sleep. [41]

Uremic toxins are retention compounds resulting in uremic syndrome. To be considered as a uremic toxic compound following criteria should be fulfilled.

(1) It should be possible to identify it and analyse it quantitatively in body fluids.

(2) It should have higher levels in plasma in uremic subjects than in nonuremic subjects.

(3) High concentrations should be related to symptoms that decrease when the concentration is reduced.

(4)These changes should be demonstrated in in-vivo, ex-vivo, or in-vitro studies; and

(5) the concentrations in these studies should resemble the concentrations present in body fluids of uremic subjects.

Urea has been used as a marker of uremic retention for many years now. It is also a surrogate marker for removal of other solutes which seem to have more established toxic effect.

One of the most important of these is potassium, a small-water soluble compound known to substantially affect survival. Another possibility is retention of other uremic solutes with. Lastly, urea is the origin of other more toxic entities including guanidines or carbamylation products

SEPSIS

Sepsis can be secondary to infections of skin/soft tissue/lung/endocarditis in order of percentage share. Pandey et al used qSOFA score to diagnose patients with sepsis in pyelonephritis and found it was a reliable bedside test with high predictive accuracy. [42]

Septic encephalopathy is defined as diffuse cerebral dysfunction due to the dysregulated host response and absence of a direct central nervous system (CNS) infection. Septic associated encephalopathy is the most common cause for encephalopathies in ICUs worldwide affecting up to 50% of patients during the course of sepsis.[43] Eidelman et al reported that the severity of neurological symptoms secondary to encephalopathy in patients in the ICU, as assessed by the Glasgow Coma Scale, was correlated to prognosis, with a mortality rate of up to 63% in patients who presented with Glasgow Coma Scale scores between 3 and 8.[44]

There is no consensual definition for septic encephalopathy. It is often a diagnosis of exclusion. Tong et al defined patients to have sepsis associated encephalopathy if they satisfied 2 of the following criteria.

1) Patient in sepsis with GCS less than 15/Abnormal EEG

2) Clinical presentation as more than 1 organ dysfunction.

3) CNS dysfunction with extracranial organ dysfunction

4) If a pure CNS dysfunction is present then CSF analysis to rule out meningitis/encephalitis.[45]

In another study from China, Zhang et al. retrospectively reviewed patients admitted to ICU over a 3-year period. The exclusion criteria included pre-existing or chronic liver or kidney failure, severe electrolyte imbalances, blood glucose disturbances, CNS infections or pre-existing CNS disease, previous cardiac arrest with resuscitation or previous treatment with

sedative drugs. Out of 323 patients, 41 had SAE and the rest had sepsis without encephalopathy. This analysis demonstrated that patients with higher APACHE II (Acute Physiology and Chronic Health Evaluation II) scores and lower scores on the Glasgow coma scale (GCS) are more likely to have SAE. Patients with SAE had significantly higher heart rate, blood lactate and serum sodium levels, as well as lower platelet count, serum albumin levels and serum pH than patients without sepsis associated encephalopathy.[46]

In a study by Chen et al it was found that hypertensive patients had more risk of progression to septic encephalopathy.[47]

Andrea polito et al conducted prospective observational study on septic shock patients. MRI showed cerebral infarcts in 21 patients (29%), leukoencephalopathy in 15 patients (21%), and mixed lesions in 6 patients (8%). Malignant pattern of EEG was more frequent in patients with ischemic stroke or leukoencephalopathy.[48]

HYPERCALCEMIA

The occurrence of acute kidney injury in hypercalcemia is secondary to a decrease of glomerular ultrafiltration coefficient, the induction of nephrogenic Diabetes insipidus via down-regulation of aquaporin-2, disruption of counter current multiplier system and a loop diuretic-like effect , that leads to polyuria and volume depletion.[49] [50] [51]

A study by Jerry et al observed that in patients with acute kidney injury for less than 48 h, the EEG was grossly abnormal and there were elevated levels of PTH in plasma. The PTH appeared to have a direct effect on the brain, resulting in an increased brain Ca^{++} content.[52]

In a study by Mousseaux et al 51% of patients with severe hypercalcemia had neurological features of which major manifestation was delirium[53]

Hypercalcemia has been found to be associated with prolonged psychosis including depression, bizarre behaviour patterns and hallucinations.[54]

In a case report by Van den Hauwe a 50 year old male with hypercalcemia presented with fatigue, depression, thirst and polyuria.[55]

The risk of neurological complications has been found to correlate with 2 factors-higher calcium on day 1 and underlying solid organ malignancy.[53]

TROPICAL FEVER

Tropical fevers form a major share of Acute kidney injury in developing countries especially India.[56]

Shukla et al found that 21.8% of patients with malaria associated Acute kidney injury presented with neurological features. [57] In a study by Naqvi et al 41% of patients with malaria associated acute renal failure had central nervous system symptoms and 81% of patients with central nervous system symptoms expired as compared to 29 % who survived thereby proving Central nervous system abnormalities to be important predictor of mortality.[58]

Neurological complications of dengue virus infection were classified into three categories based on pathogenesis as proposed by Murthy, Marzia and colleagues:

(1) Metabolic disturbance, e.g., encephalopathy.

(2) Viral invasion, including encephalitis, meningitis, myositis, and myelitis; (3) Autoimmune reactions, including acute disseminated encephalomyelitis, neuromyelitis optica, optic neuritis, myelitis, encephalopathy, and Guillain-Barré syndrome.[59]

Encephalopathy caused by dengue fever can be seen as reduced sensitivity, cognitive impairment, convulsions, and personality and

behaviour disorders, including acute mania, depression, emotional lability, anxiety, psychosis, and agoraphobia.[60]

A Study was done by Diptyanusa et al over a period of 5 years and included 1484 patients with dengue. In this study the prevalence of acute kidney injury was 4.8 %. It also observed that almost all patients who developed altered levels of consciousness belonged to the acute kidney injury group (incidence was 0.1% and 15.3% in non-acute kidney injury and acute kidney injury groups, respectively). Such altered consciousness in patients could have resulted from shock, electrolyte disturbances, or uremic encephalopathy.[61]

Encephalitis is the most common neurological manifestation of dengue infection, and the main symptoms include seizures, altered consciousness, and headaches. In A study done by Jackson et al clinical manifestations in 54 dengue positive with CNS manifestations included encephalitis in 28 patients(51.8%), meningitis in 18 patients(33.3%), seizures in 6 patients [1 with encephalitis, 1 with paralysis] (11.1%), AFP/GBS in 2 patients(3.7%).[62]

The diagnosis criteria for dengue encephalitis consists of: fever and acute signs of cerebral involvement associated with the presence of anti-dengue IgM or dengue RNA in the serum and/or CSF added to the exclusion of other causes of viral encephalitis and encephalopathy.[63] In these cases computed tomography (CT) and magnetic resonance imaging (MRI) findings may be normal.[64]

In acute phase of dengue infection, dengue shock syndrome (DSS), brain oedema, cerebral anoxia, metabolic acidosis, electrolyte disturbances, vasculitis, liver and kidney failure can contribute to encephalopathy.[65]

A Study conducted by Kalita et al included 24 patients with dengue virus. EEG revealed theta to delta slowing in 11 out of 20 patients. Five of these patients had seizures, 12 patients had altered sensorium, 3 patients had

hypotension, 9 patients had CSF pleocytosis, 3 patients had moderate to severe liver dysfunction and 2 patients had MRI abnormalities.[66]

In a study done by Khalil et al on 532 patients with dengue, acute kidney injury was present in 71 patients (13.3%). Neurological involvement (OD 12.08; 95% CI 2.82–51.77) was found to be an independent predictor for development of acute kidney injury.[67]

R.A. Fearnley et al reported a case of Influenza A-induced rhabdomyolysis causing Acute kidney injury in a young adult which was further complicated by posterior reversible encephalopathy syndrome.[68]

A cross sectional study conducted by Misra et al on 37 patients with scrub typhus and neurological changes showed Thirty-one (84%) patients had impaired consciousness, and six patients were deeply comatose (GCS score ≤8). Eight patients presented with status epilepticus. MRI was done in 25 patients and revealed meningeal enhancement in only 1 patient (4%) patient. EEG was done in 28 patients and showed generalised slowing in 6 patients (21.4%). Among 31 patients with altered sensorium, CSF studies were conducted on 28. 19 patients had meningoencephalitis and 9 patients had encephalopathy.[69]

NEPHROTOXIN

Sam Alfred et al observed in a study on ethylene glycol poisoning that out of 7 patients with anuric acute renal failure 3 patients developed significant cranial neuropathies with bulbar palsy in the second week post ingestion. One patient died with cerebral oedema and a progressive encephalopathy. 2 patients with normal renal function and a moderate metabolic acidosis had no neurological sequalae.[70]

The pharmacokinetics of drugs undergo change in renal injury hence neurological side effects of drugs are expected to be precipitated in Acute kidney injury.

D Chatellier et al reported 5 patients suffering from acute renal failure who were treated with cefepime and presented with seizures and coma.[71]

HEPATIC ENCEPHALOPATHY (HE)

Hepatic encephalopathy (HE) is a reversible syndrome of impaired brain function occurring in patients with advanced liver disease. Its onset may be gradual or sudden. The pathogenesis of HE includes a complex network which remains poorly characterized. is a frequent complication of chronic liver disease. Its pathogenesis is not understood. There is nonetheless a growing recognition that there is a complex but influential synergistic relationship between ammonia, inflammation and oxidative stress in the pathogenesis HE.[72]

Cerebral oedema and increased intracranial pressure can occur in chronic liver disease and present as neurological deterioration.[73]

Episodes of HE can be triggered by infections, gastrointestinal bleeding, constipation, electrolyte problems especially hypokalaemia or medications especially sedative drugs. The diagnosis is typically based on symptoms after ruling out other potential causes.

HE may present as anxiety or irritability, cognitive impairment (confused thinking or judgment), coordination or balance problems, difficulty concentrating or short attention span, flapping hand motion (asterixis), mood or personality changes or as muscle twitches (myoclonus).

Three types of HE is differentiated according to the underlying cause: Type A as a component of acute liver failure, type B as a consequence of Porto-systemic shunts in the absence of liver dysfunction and type C in patients with liver cirrhosis.[74] Several scales have been used to grading the extent of HE.

The most often used is the West Haven criteria (WHC), which differentiate between four grades of clinically overt HE. Patients with liver cirrhosis

without clinically overt symptoms of HE but neuropsychological or neurophysiological findings indicating brain dysfunction are considered to have minimal hepatic encephalopathy (MHE). MHE is the earliest form of hepatic encephalopathy and can affect up to 80% of cirrhotic patients but it has no obvious clinical manifestation.[75] The overall prevalence of HE in cirrhosis is 30 – 45 %.[76]

HUS is the most common cause of Acute kidney injury in children but is increasing being diagnosed in adults. In a study on patients with HUS Nathanson et all found pts had stuporous coma, protracted severe seizures, and other neurologic defect including pyramidal syndrome, hemiplegia or hemiparesis, extrapyramidal syndrome and coma.[77] Steinborn et al studied 57 patients with clinically proven HUS. Of these 57 patients in 6 patients pathological changes were found on CT and MRI within the basal ganglia.[78] Karin Weissenborn found that among 48 patients of HUS All but 1 showed neurologic symptoms. Focal neurologic signs like double vision, difficulties in finding words, or hyperreflexia were present in 23 patients, additional deficits in orientation, attention, memory, or constructive abilities were present in 9 patients, and marked impairment of consciousness was found in 15 patients. MRI showed brainstem, midbrain, thalamus, corpus callosum, and white matter lesions in half of the patients, predominantly in diffusion-weighted images. General slowing was found in half of the patients examined by EEG.[79]

Grażyna Zarzycka-Lindner, et al reported a case of 42-year-old man who developed symptoms of Guillain-Barre syndrome (GBS) 5 days after development of acute kidney injury.

Wairagkar et al reported manifestations of acute renal failure with neurological involvement (bilateral facial weakness, impaired hearing, blindness, proximal and distal areflexic limb paralysis, and respiratory paralysis) associated with measles virus in adults.[80]

MATERIAL AND METHODS

A prospective Observational Study was Conducted on Dayanand Medical College and Hospital, Ludhiana Which Included 200 Patients Diagnosed with Acute kidney injury.

Inclusion Criterion:

1. All Patients >18 Years of either gender admitted in DMCH and diagnosed with Acute kidney injury were included in the study.

Exclusion Criterion:

Following patients were excluded from the study.

- Patients With Underlying Chronic Kidney Disease as Defined by KDIGO Criteria
- Post Renal Transplant
- Patients On Anti-Epileptic Maintenance Treatment
- Patients On Mood Stabilizers, Anti-Depressants, Antipsychotics
- Survivors Of Brain Tumours
- Patients With History of Neurosurgical Procedures.

Number of Cases: 200 patients satisfying the inclusion criteria were included in the study.

METHOD OF COLLECTION OF DATA

Comprehensive clinical examination was documented including GCS along with blood counts, chemistries, and other relevant investigations on a proforma. History of Nephrotoxins was elicited verbally or with documented medical records of patients. This included poisoning and other therapeutic drugs with possible nephrotoxic side effect including antibiotics. Poisoning was attributed as aetiology of AKI when the renal

deterioration occurred secondary to the drug effect alone. As the SOFA score is not universally accessible qSOFA score has been proposed to assess patients bedside clinically for risk of deterioration due to sepsis in wards, emergency department outside the intensive care unit.[42] [81] In our study patients were defined to have sepsis if qSOFA score was more than or equal to 2 and clinical evidence of infection was present. qSOFA score includes a)RR ≥22/min b)SBP <100 mmHg c)altered mentation, defined as reduction in GCS (GCS <15).[81] Patients were defined to be in septic shock if inotropes were required to maintain MAP at or above 64 mmHg and lactate levels -were more than 2.[81] Renal replacement included C.R.R.T, peritoneal dialysis , haemodialysis and S.L.E.D. Acute kidney injury was defined using KDIGO criteria (2012): increase in serum creatinine by more than or equal to 0.3 mg/dL within 48 hours or increase in serum creatinine to 1.5 times of baseline or more within last 7 days or urine output less than 0.5 ml/kg/hr for 6 hours or more.[1] Staging of acute kidney injury was done to classify patients in stage 1, 2 and 3 using KDIGO criteria 2012.[1]

Stage	**Serum creatinine**	**Urine output**
1	1.5–1.9 times baseline OR more than or equal to 0.3 mg/dl increase.	Less than 0.5 ml/kg/h for 6–12 hours.
2	2.0–2.9 times baseline	Less than 0.5 ml/kg/h for 12 hors or more.
3	3.0 times baseline OR Increase in serum creatinine to more than or equal 4.0 mg/dl OR Initiation of renal replacement therapy.	Less than 0.3 ml/kg/h for more than 24 hours OR Anuria for 12 hours or more.

- Neurological changes were assessed by baseline scores and neurological examination was done on day 0 and day 7 of admission.

- Patients with GCS score less than 15 were classified as having neurological complication.
- MMSE was applied on patients with GCS score of 15.
- Richmond agitation sedation scale was used to assess level of sedation.
- Patients were screened for delirium by using CAM ICU score. The CAM-ICU is modified from the Confusion Assessment Method (CAM) and assesses four features: 1) Acute change or fluctuation in mental status from baseline, 2) inattention, 3) altered level of consciousness and 4) disorganized thinking.[82] The CAM-ICU is positive, and the patient is considered to have delirium, if Features 1 and 2 and either Feature 3 or 4 is present.
- 9 patients were lost to follow up on day 7(Left against medical advice).

Outcome:

The primary Outcome of study was to see the Spectrum of Neurological Changes in Acute kidney injury.

The secondary outcome was to assess any correlation in neurological severity in progressive stages of Acute kidney injury.

Statistical Analysis:

Data has been described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (%s) as appropriate. All statistical calculations were done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

OBSERVATION AND RESULTS

AGE AND SEX

Table 1: Age distribution of patients with acute kidney injury

Age group	No. of cases	%
< 30	26	13.0%
31-40	18	9.0%
41-50	25	12.5%
51-60	69	34.5%
61-70	51	25.5%
> 70	11	5.5%
Total	200	100.0%

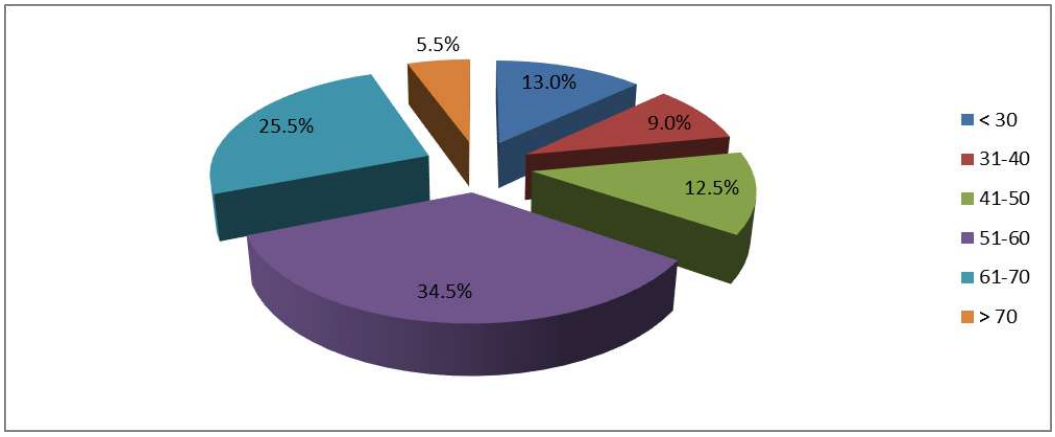


Figure 1 : Age distribution of patients with acute kidney injury

In our study 200 patients were studied. Of these maximum percent of patients were in 6th decade (34.5%). Mean age of patients was 55.9 years. Patients above the age of 50 years were more likely to develop neurological complications however p value was not significant.

Table 2 : Gender distribution of patients with Acute kidney injury

Sex	No. of cases	%
F	84	42.0%
M	116	58.0%
Total	200	100.0%

116 patients (58 %) were males, and 84 patients (42 %) were females. There was no significant correlation between sex of patients and development of neurological complications.

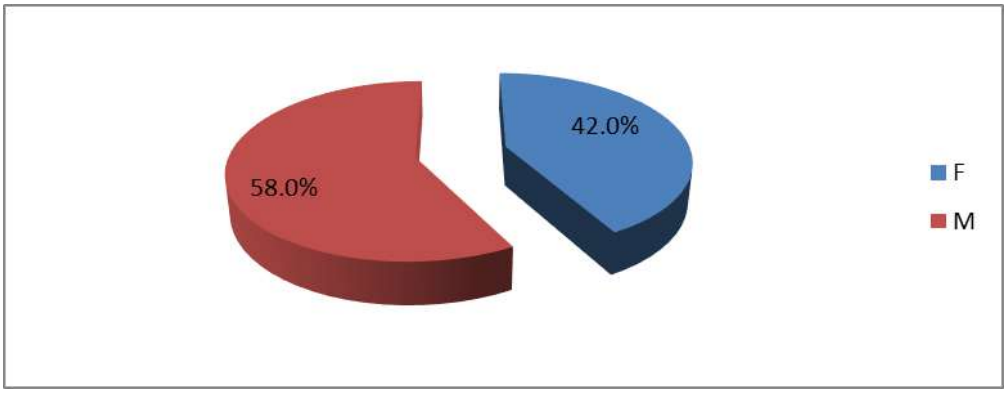


Figure 2 : Gender distribution of patients with Acute kidney injury

COMORBIDITIES

Table 3: Comorbidities in patients with Acute kidney injury

	No. of cases	%
Hypertension	68	34.0%
Diabetes mellitus 2	68	34.0%
CLD	16	8.0%
CAD	32	16.0%

68 patients (34 %) had previous history of Hypertension, 68 patients (34 %) had Diabetes mellitus 2, 32 patients (16 %) had previously diagnosed CAD, 16 patients (8 %) had previously diagnosed chronic liver disease.

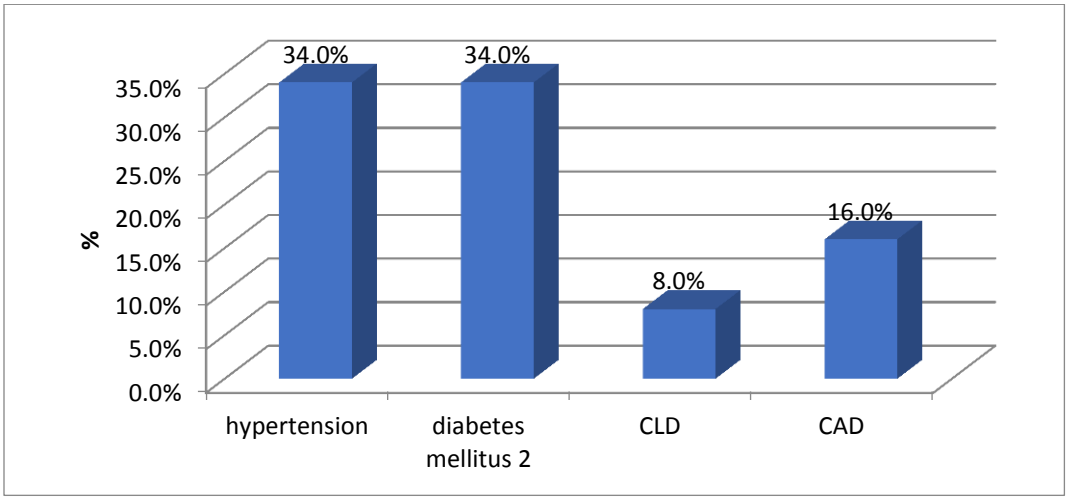


Figure 3 : Comorbidities in patients with Acute kidney injury

Table 4 : Influence of comorbidities on development of neurological features

		NEUROLOGICAL FEATURES yes/no				Total	Chi-square value	p-value
		N	N	Y	Y			
Hypertension	N	109	69.9%	23	52.3%	132	4.737	0.046
	Y	47	30.1%	21	47.7%	68		
Diabetes mellitus 2	N	98	62.8%	34	77.3%	132	3.194	0.104
	Y	58	37.2%	10	22.7%	68		
CLD	N	143	91.7%	41	93.2%	184	0.107	0.744
	Y	13	8.3%	3	6.8%	16		
CAD	N	128	82.1%	40	90.9%	168	2.004	0.157
	Y	28	17.9%	4	9.1%	32		

In our study Patients with underlying Hypertension were more likely to have neurological complications as compared to non-hypertensive patients with a significant p value. No correlation was found between patients who developed neurological complications and patients with underlying Diabetes mellitus, coronary artery disease and chronic liver disease.

SUBSTANCE ABUSE

Table 5: Substance abuse in patients with acute kidney injury

H/O Alcohol/smoking/substance abuse	No. of cases	%
No	158	79.0%
Yes	42	21.0%
Total	200	100.0%

21 % of patients had history of substance abuse including alcohol, opioid and other intravenous drugs.

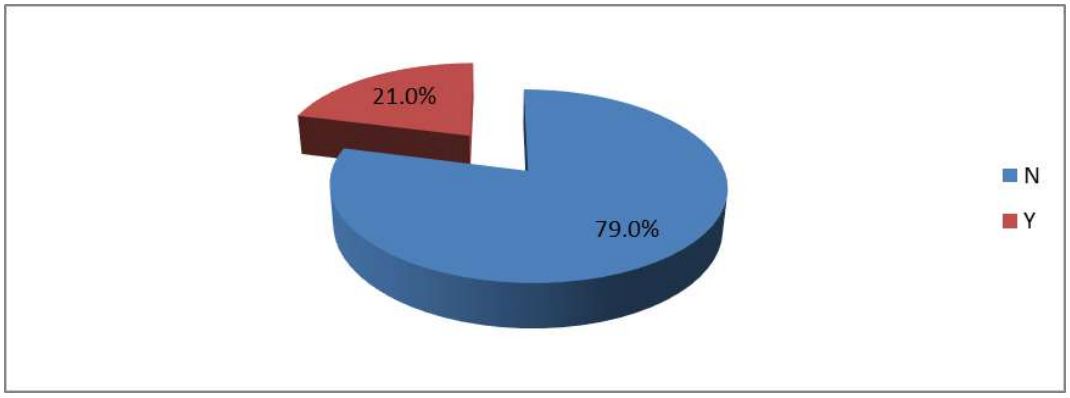


Figure 4 : Substance abuse in patients with acute kidney injury

NEPHROTOXIC DRUGS AND POISONINGS

Table 6A : Nephrotoxic drugs history in patients with acute kidney injury.

	No. of cases	%
Nephrotoxic drugs prior to admission	16	8.0%

16 patients (8 %) had history of nephrotoxic drugs which included antibiotics-vancomycin, colistin, amikacin; NSAIDS; Poisoning- 5 patients had history of paraquat intake, and 1 patient had history of celphos ingestion. 2 patients had inadvertent intake of vitamin d which caused hypercalcemia leading to acute kidney injury. 2 patients had history of opioid intake mixed with herbal products, 1 patient had history of alternative medication.

Table 6B: Type of nephrotoxin in patients of Acute kidney injury

NEPHROTOXIC DRUG	NUMBER OF PATIENTS
ANTIBIOTIC	3
NSAIDS	2
POISONING	6
VITAMIN D	2
ALTERNATIVE MEDICATION	1
MISCELLENAEOUS-	2
TOTAL	16

ICU STAY

Table 7 : Patients with acute kidney injury requiring ICU care, ventilatory support and inotropic support.

	NUMBER OF PATIENTS (200)	% OF TOTAL PATIENTS
ICU	67	33.5
VENTILATORY SUPPORT	9	4.5
IONOTROPIC SUPPORT	40	20

67 patients (33.5 %) had to be admitted to ICU. 9 patients (4.5%) required mechanical ventilatory support and 40 patients (20 %) needed ionotropic support.

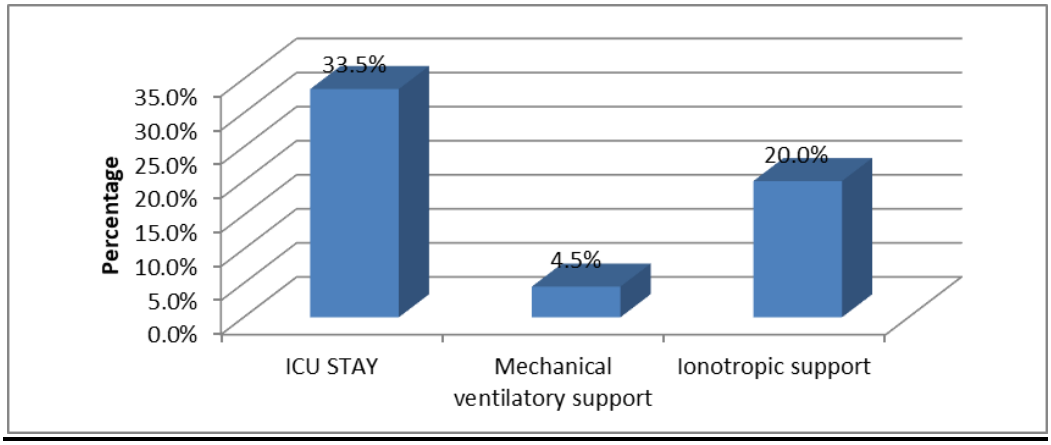


Figure 5 : Patients with acute kidney injury requiring ICU care, ventilatory support and ionotropic support.

Table 8: Correlation of need for ICU and development of neurological features.

		NEUROLOGICAL FEATURES yes/no				Total	Chi-square value	p-value
		N		Y				
ICU STAY	N	124	79.5%	9	20.5%	133	53.687	0.001
	Y	32	20.5%	35	79.5%	67		

Patients who required intensive care unit admission were more likely to have developed neurological features with a significant p value of 0.001.

Table 9: Correlation of need for mechanical ventilation and ionotropic support with development of neurological features.

		NEUROLOGICAL FEATURES yes/no				Total	Chi-square value	p-value
		N		Y				
Mechanical ventilatory support	N	156	100.0%	35	79.5%	191	33.413	0.001
	Y	0	0.0%	9	20.5%	9		
Ionotropic support	N	135	86.5%	25	56.8%	160	18.947	0.001
	Y	21	13.5%	19	43.2%	40		

Patients who required ventilatory support were more likely to have developed neurological features with significant p value of 0.001.

Patients who required ionotropic support were more likely to have developed neurological features with significant p value of 0.001

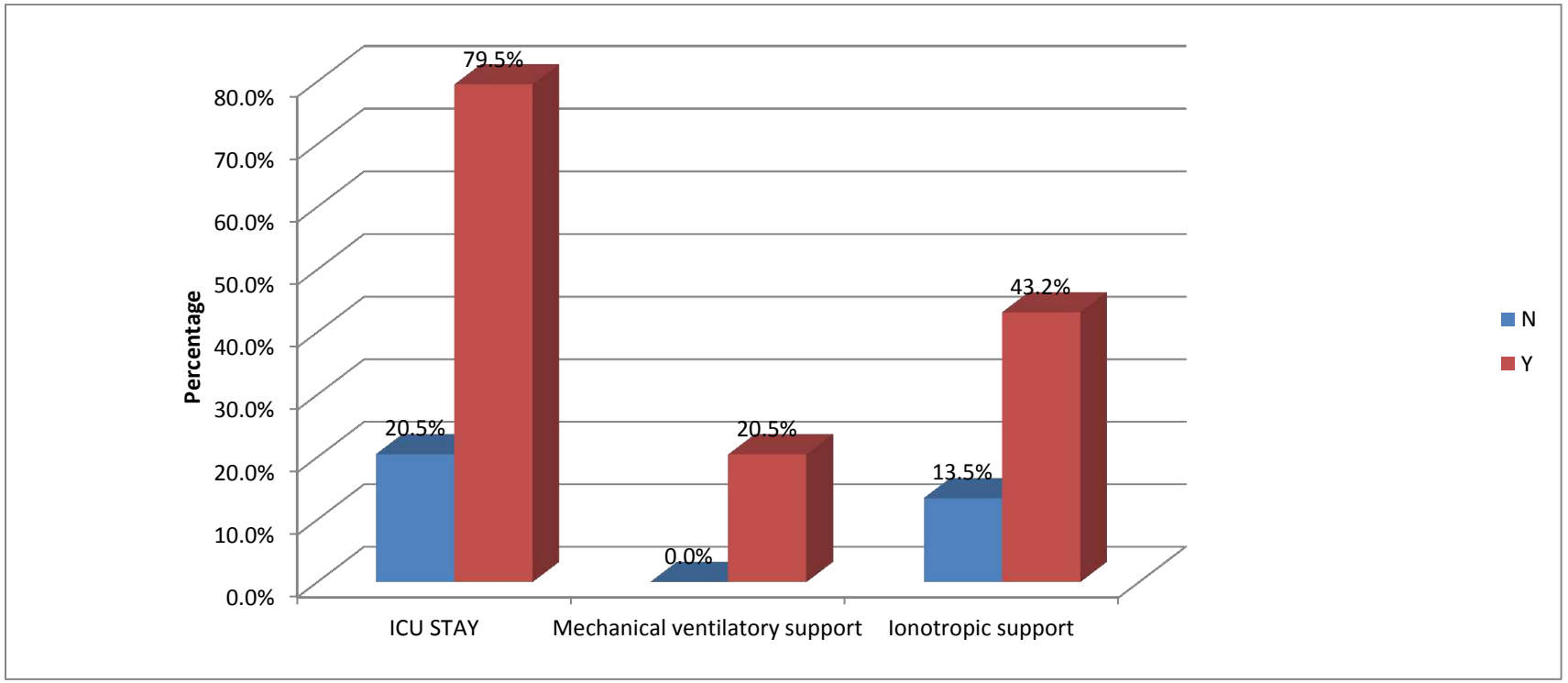


Figure 6: Correlation of need for mechanical ventilation and ionotropic support with development of neurological features

ETIOLOGY OF ACUTE KIDNEY INJURY

Table 10A: Aetiology of acute kidney injury

CAUSE	No. of cases	%
CARDIORENAL	10	5.0%
HRS	2	1.0%
HYPOVOLUMEA	3	1.5%
INFECTION	117	58.5%
MISCELLENEOUS	22	11.0%
NEPHROTOXIN	9	4.5%
OBSTRUCTIVE	2	1.0%
PANCREATITIS	6	3.0%
SURGICAL	7	3.5%
TROPICAL FEVER	22	11.0%
Total	200	100.0%

The most common cause of Acute kidney injury was infections which accounted for 119 patients-59 % of patients followed by tropical fever which accounted for 22 patients (11%) and miscellaneous causes including hypercalcemia, ANCA vasculitis, polymyositis. Less common causes include HRS, Obstructive, and hypovolemia. The most common site for infection was gastrointestinal followed by urosepsis and thirdly respiratory.

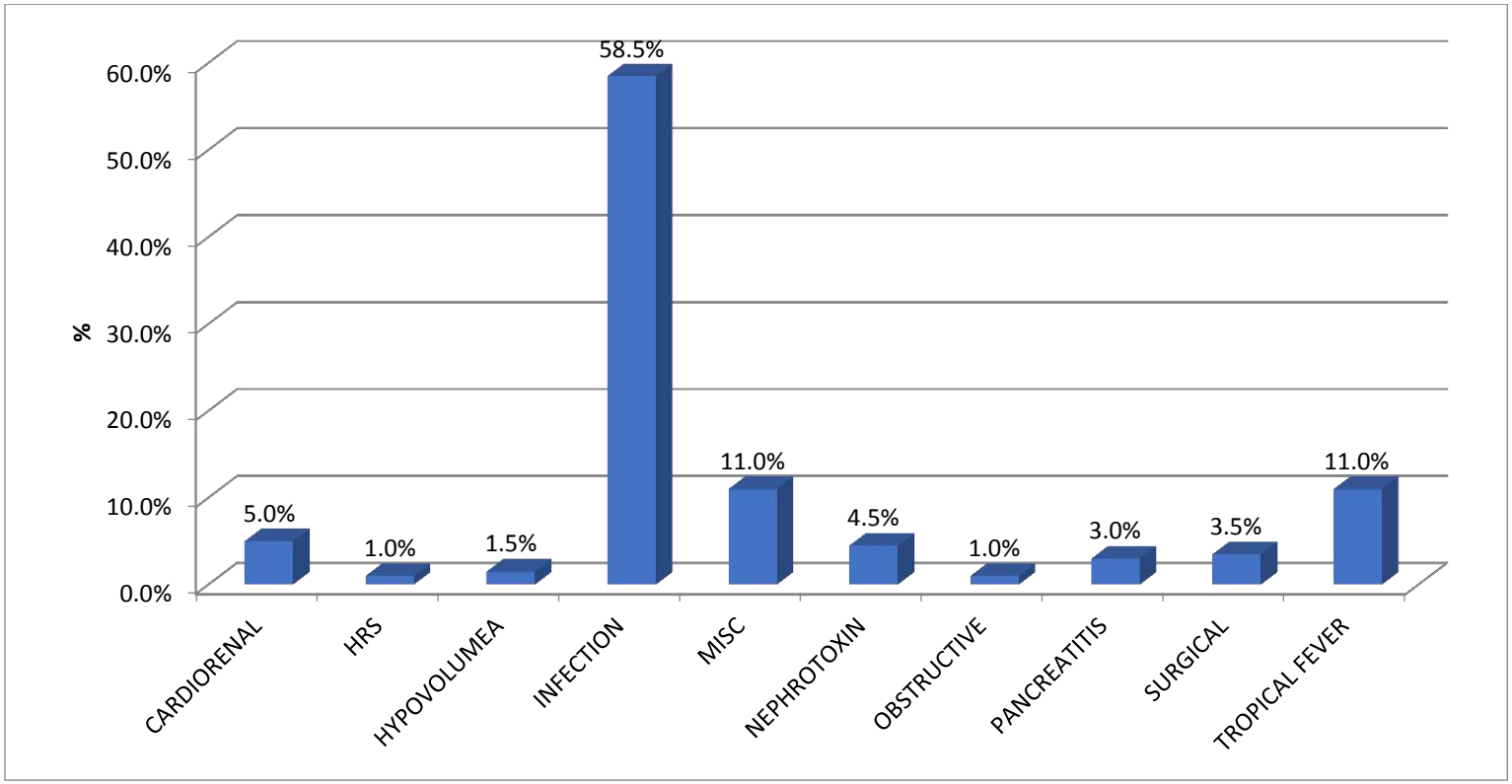


Figure 7: Aetiology of acute kidney injury.

Table 10B: Causes of acute kidney injury.

	No. of cases	%
CARDIORENAL	10	5.0%
ACS	9	4.5%
HYPERTENSIVE URGENCY	1	0.5%
HRS	2	1.0%
HYPOVOLUMEA	3	1.5%
INFECTION	117	58.5.0%
GASTROINTESTINAL AND HEPATOBILIARY	43	21.5%
BACTEREMIA	1	0.5%
SKIN AND SOFT TISSUE	4	2.0%
RESPIRATORY	21	10.5%
INFECTIVE ENDOCARDITIS	2	1.0%
CNS AND OCULAR	4	2.0%
UROSEPSIS	37	18.5%
MISC AND MULTIPLE	5	2.5%
MISCELLENEOUS	22	11.0%
ANCA VASCULITIS	1	0.5%
DIABETIC KETOACIDOSIS	3	1.5%
DISSEMINATED TUBERCULISIS, POLYMYOSITIS	1	0.5%
HEMOLYTIC UREMIC SYNDROME	1	0.5%
HIV	1	0.5%
HYPERCALCEMIA	6	3.0%
IgG4 DISEASE	1	0.5%
MALIGNANCY-SOLID ORGAN OR HEMATOLOGICAL	4	2.0%

SNAKE BITE	3	1.5%
POISONING	9	4.5%
OBSTRUCTIVE	2	1.0%
HYDROURETRONEPHROSIS	1	0.5%
NEPHROLITHIASIS	1	0.5%
PANCREATITIS	6	3.0%
SURGICAL	7	3.5%
ECTOPIC PREGNANCY	1	0.5%
INTESTINAL OBTSRUCTION	6	3.0%
TROPICAL FEVER	22	0.0%
COMPLICATED TROPICAL FEVER	2	1.0%
DENGUE	12	6.0%
LEPTOSPIRA	4	2.0%
LEPTOSPIRA AND SCRUB TYPHUS	1	0.5%
MALARIA FALCIPARUM	1	0.5%
SCRUB TYPHUS	2	1.0%

The most common source for infections was gastrointestinal-hepatobiliary followed by urinary tract infection and respiratory infection.

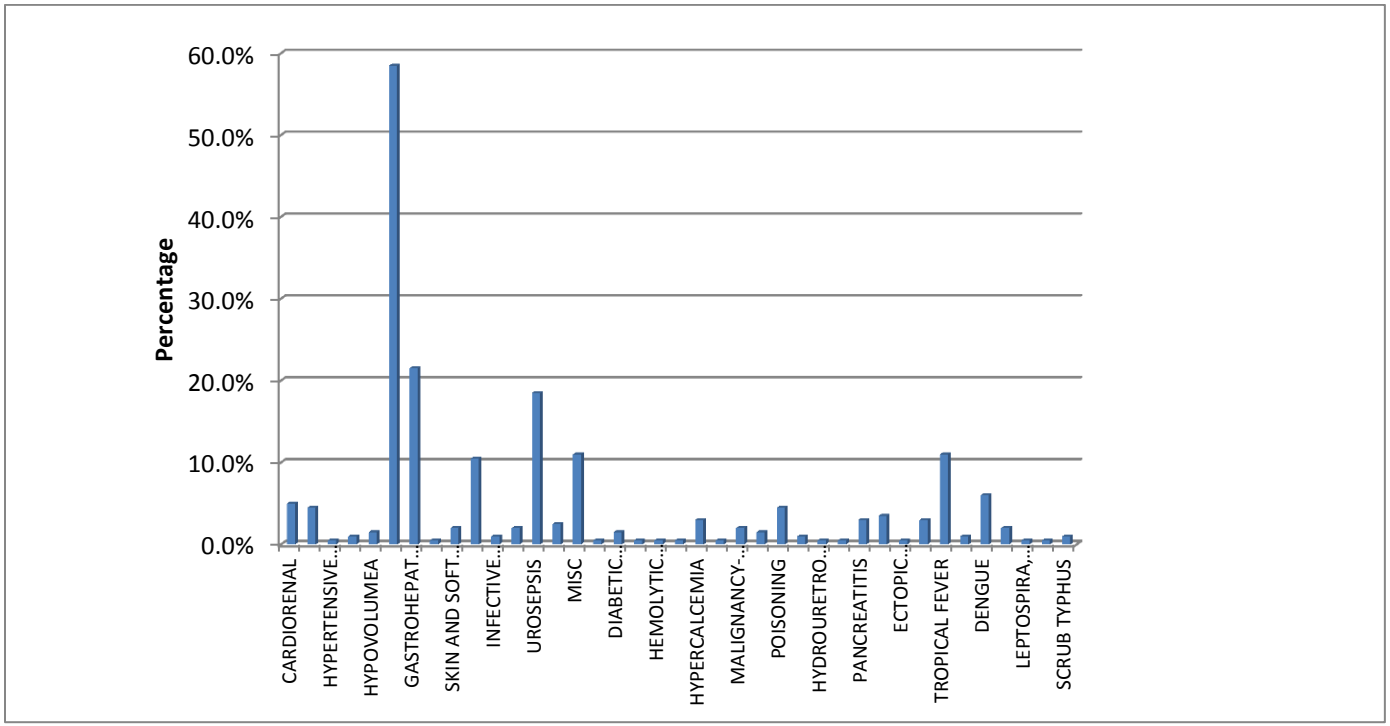


Figure 8: Causes of acute kidney injury.

SEPSIS AND SEPTIC SHOCK

Table 11A: qSOFA score in patients with acute kidney injury.

qSOFA _Day 0	No. of cases	%
< 1	156	78.0%
> 2	44	22.0%

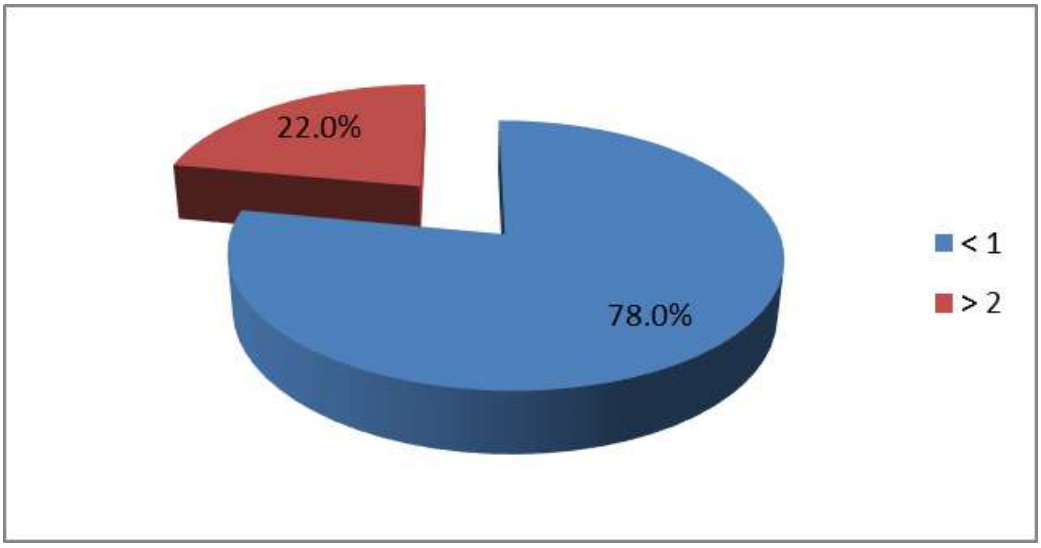


Figure 9: qSOFA score in patients with Acute kidney injury.

In our study 156 patients (78 %) had qSOFA score less than or equal to 1 while 44 patients (22 %) had qSOFA score more than equal to 2.

Table 11B : qSOFA score of patients.

qSOFA _Day 0	No. of cases	%
0	112	56.0%
1	44	22.0%
2	36	18.0%
3	8	4.0%
Total	200	100.0%

qSOFA was 0 in 112 patients which constituted 56 % of total patients.44 patients had qSOFA score 1, 36 patients (18%) had qSOFA score 2 and 8 patients had qSOFA score 3.

Table 12A: Sepsis in patients with acute kidney injury.

sepsis present (qSOFA more than 2 and evidence of infection)	No. of cases	%
N	173	86.5%
Y	27	13.5%
Total	200	100.0%

In our study sepsis was present in 27 patients of 200 patients which constituted 13.5 % of total patients.

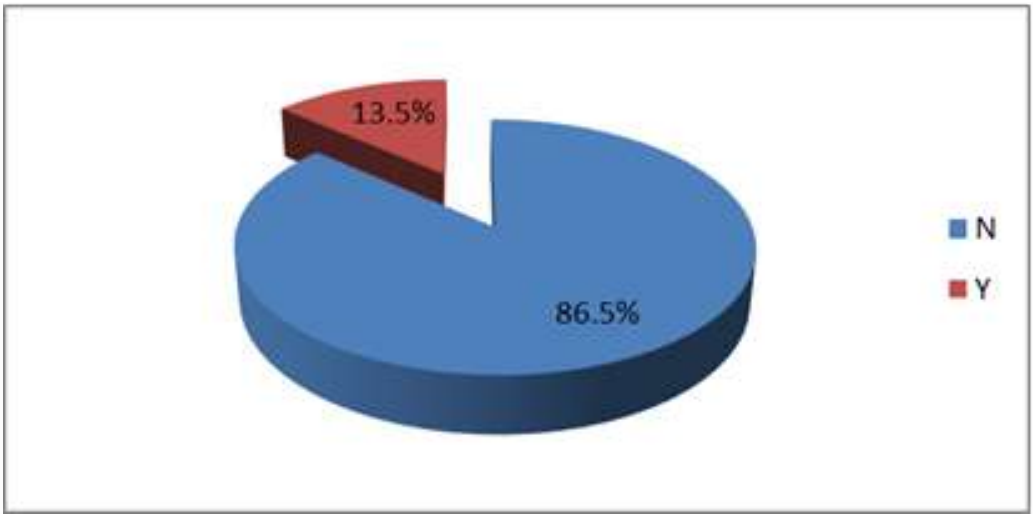


Figure 10A: Sepsis in patients with acute kidney injury.

Table 12B: Source of sepsis

Source of sepsis	No. of cases	% Of total patients	% Of sepsis
BACTEREMIA	1	0.5%	3.7%
CELLULITIS	1	0.5%	3.7%
DIABETIC FOOT	2	1.0%	7.4%
ECTOPIC PREGNANCY	1	0.5%	3.7%
GASTROHEPATOBILARY	5	2.5%	18.5%
INFECTIVE ENDOCARDITIS	1	0.5%	3.7%
ORBITAL	1	0.5%	3.7%
RESPIRATORY	5	2.5%	18.5%
PSOAS ABCESS	1	0.5%	3.7%
TROPICAL FEVER	1	0.5%	3.7%
UROSEPSIS	8	4.0%	29.8%

The most common source of sepsis in our study was urosepsis which accounted for 29 % of patients with sepsis and 4 % of total 200 patients. This was followed by respiratory site presenting as pneumonia in 18.5% of patients with sepsis and 2.5% of total patients along with gastrohepatobiliary which also accounted for 18.5 % of patients with sepsis and 2.5 % of total patients. Other sources were cellulitis, diabetic foot, ectopic pregnancy, infective endocarditis, meningitis, orbital cellulitis, psoas abscess, tropical fever which included-dengue, leptospirosis, scrub typhus and malaria.

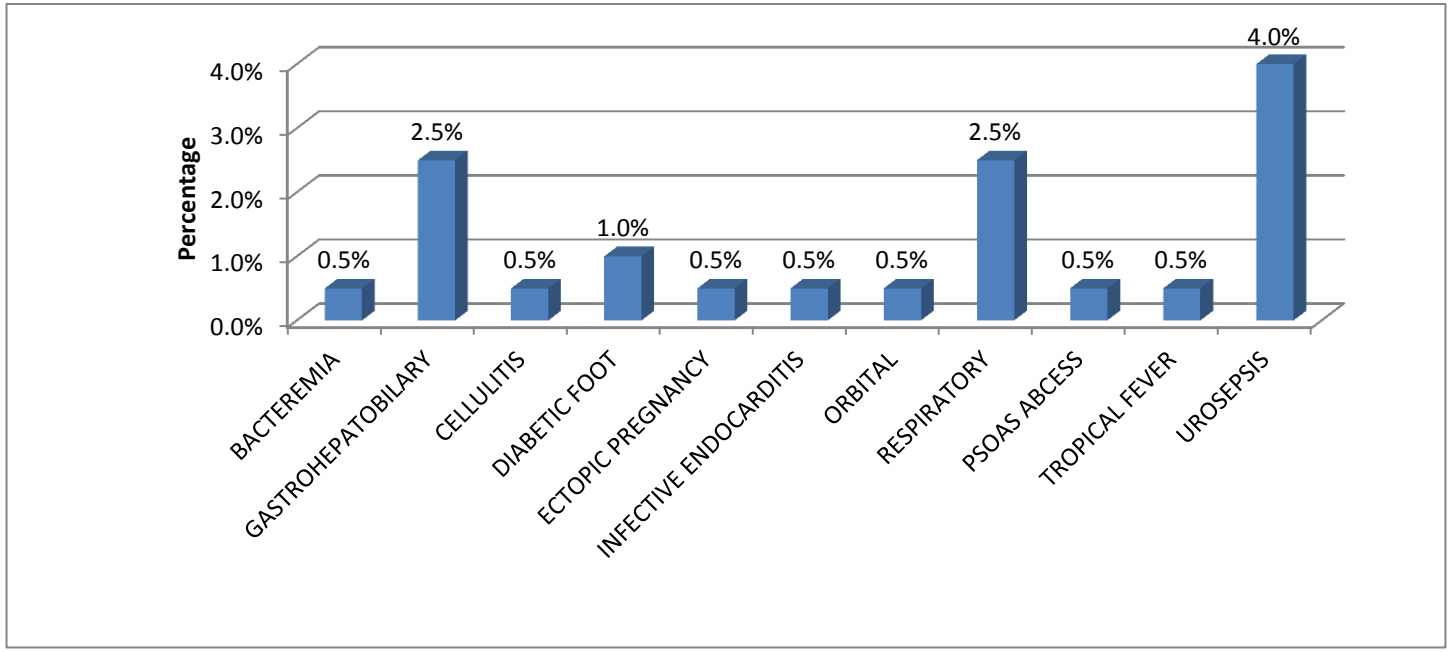


Figure 10 B: Source of sepsis

Table 13: Septic shock in patients with sepsis

Septic shock present	No. of cases with sepsis	Percentage
N	7	25.9%
Y	20	74.1%
Total	27	100.0%

In our study septic shock was present in 20 patients which constituted 74 % of patients with sepsis.

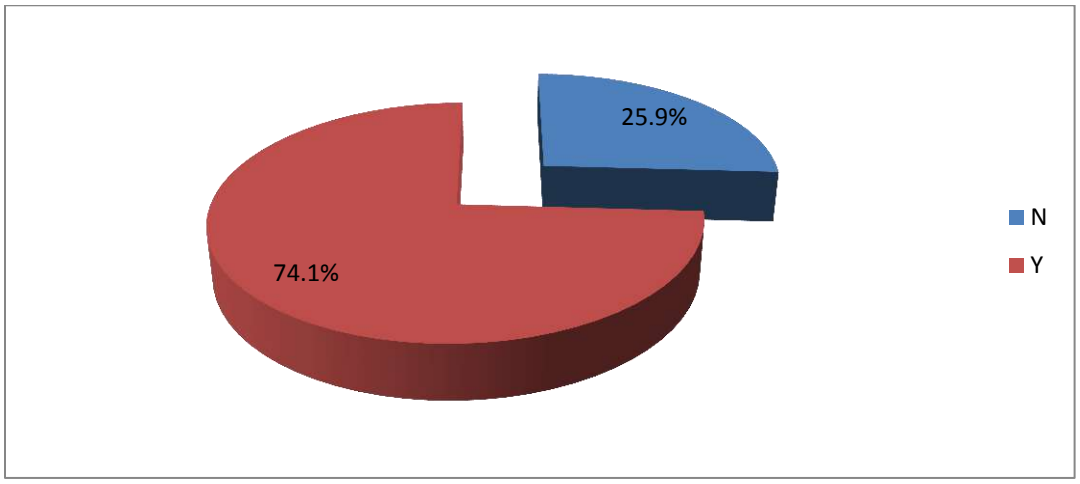


Figure 11: Septic shock in patients with sepsis.

Table 14: Influence of presence of sepsis and septic shock on development of neurological complications.

		NEUROLOGICAL FEATURES yes/no		Total	Chi-square value	p-value
		N	Y			
sepsis present	N	142	31	173	12.437	0.001
	Y	14	13	27		
septic shock present	N	147	33	180	14.103	0.001
	Y	9	11	20		

In our study significant correlation was found between patients who had neurological complications and patients who were in sepsis and septic shock.

URINE ANALYSIS AND ULTRASOUND

Table 15: Urine Analysis of patients with acute kidney injury.

Urine R/E	No. of cases	%
ABNORMAL	116	58.0%
NORMAL	84	42.0%
Total	200	100.0%

116 patients (58%) had abnormal urine routine examination which included pus cells, proteinuria, red blood cells and bacilli.

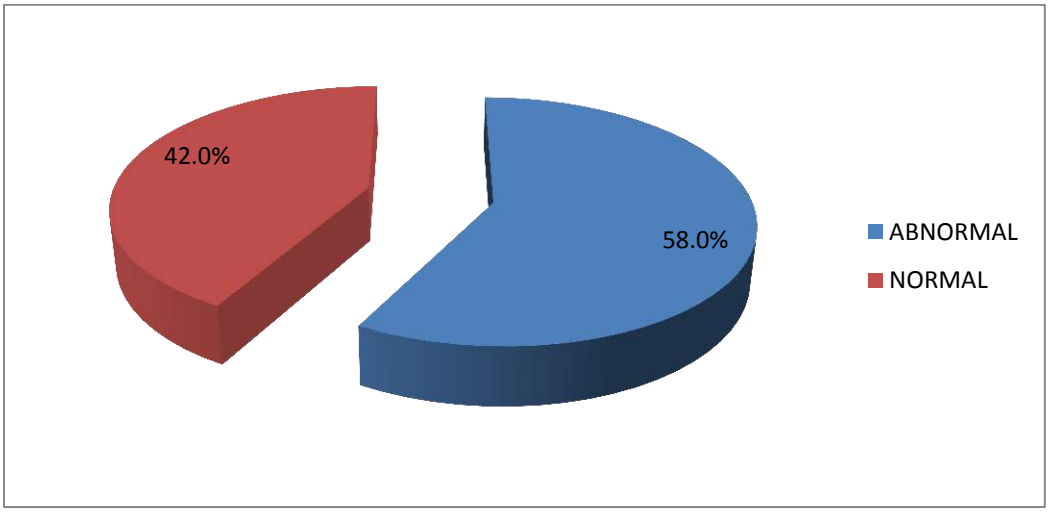


Figure 12: Urine Analysis in patients with acute kidney injury.

Table 16 : Ultrasound findings in patients with acute kidney injury.

USG abdomen	No. of cases	%
ABNORMAL	175	87.5%
NORMAL	25	12.5%
Total	200	100.0%

175 patients (87.5%) had abnormal ultrasound findings which included.

1) Gall bladder and sludge in tropical fever

2) Cystitis and pyelonephritis

3) Hepatitis

4) Chronic liver disease

5) Pancreatitis, peripancreatic stranding

6) Hepatosplenomegaly

7) Intestinal obstruction

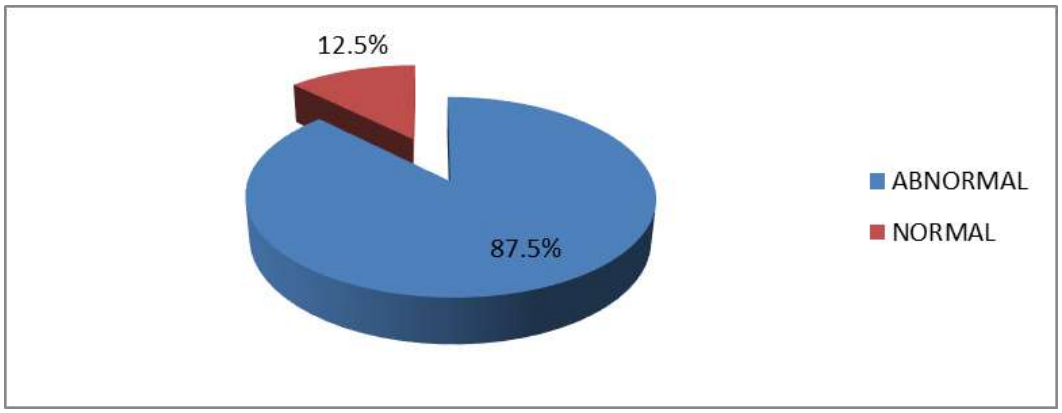


Figure 13: Ultrasound findings in patients with acute kidney injury.

CULTURE

Table 17: Urine culture in patients with acute kidney injury.

Urine culture	No. of cases	%
NOT SENT	105	52.5%
CANDIDA	2	1.0%
CANDIDA STELLOIDES	1	0.5%
E COLI	6	3.0%
ENTEROBACTER	1	0.5%
ENTEROCOCCUS	1	0.5%
ENTEROCOCCUS, SERRATIA	1	0.5%
KLEBSIELLA	3	1.5%
NO ORGANISM ISOLATED	80	40.0%
Total	200	100.0%

Urine culture was sent in 95 patients. It was sterile and did not isolate any organism in 80 patients. 15 patients had isolated organism which included Candida stelloides in 1 patient, Escherichia coli in 6 patients, Enterobacter in 1 patient, Enterococcus in 1 patient, Klebsiella in 3 patients. Enterobacter and Serratia were isolated simultaneously in 1 patient.

Table 18: Blood culture in patients with Acute kidney injury.

Blood culture	No. of cases	%
NOT SENT	106	53.0%
CANDIDA GILLIER	1	0.5%
E COLI	1	0.5%
KLEBSIELLA	1	0.5%
MRSA	1	0.5%
Normal	89	44.5%
STAPHYLOCOCCUS HEMOLYTICUS	1	0.5%
Total	200	100.0%

Blood cultures were sent in 94 patients. No organism was isolated in 89 patients. 5 patients had specific organism isolated. This included Escherichia coli in 1 patient, Candida Giller in 1 patient, Klebsiella in 1 patient, Staphylococcus Hemolyticus in 1 patient and MRSA in 1 patient.

CULTURE ISOLATES FROM OTHER SITES.

5 patients had organisms isolated in other body fluids/secretions.1 patient had Acinetobacter isolated in endotracheal secretion, 1 patient had Pseudomonas aeruginosa isolated in ascitic fluid, 1 patient had Enterococcus isolated in sputum and 1 patient had Klebsiella isolated in sputum culture and 1 patient had Proteus along with Klebsiella isolated in wound culture.

STAGING OF ACUTE KIDNEY INJURY

Table 19: AKI Stages in patients.

ACUTE KIDNEY INJURY STAGE	No. of cases	%
1	102	51.0%
2	39	19.5%
3	59	29.5%
Total	200	100.0%

KDIGO criteria (2012) was applied at time of presentation to stage patients into stage 1, 2 and 3 of acute kidney injury. 102 patients (51 %) belonged to Stage 1 Acute kidney injury, 39 patients (19.5 %) belonged to stage 2 Acute kidney injury and 59 patients (29.5 %) belonged to stage 3 Acute kidney injury.

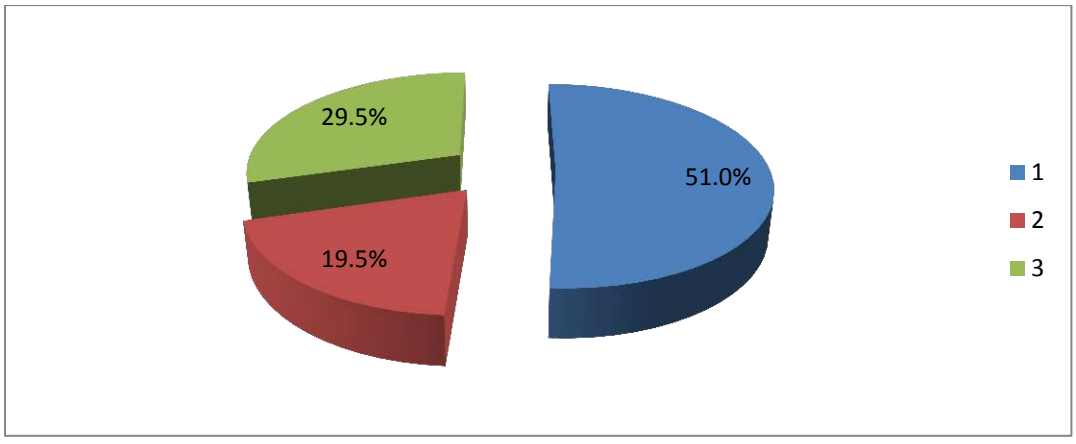


Figure 14 - Percentage of patients in stages of AKI

TABLE 20: Correlation Of Stage Of Aki With Presence Of Neurological Features

		NEUROLOGICAL FEATURES yes/no				**Total**	**Chi-square value**	**p-value**
		N		**Y**				
AKI STAGE	1	82	52.6%	20	45.5%	102	1.291	0.524
	2	31	19.9%	8	18.2%	39		
	3	43	27.6%	16	36.4%	59		
Total		156	100.0%	44	100.0%	200		

There was no correlation of staging of acute kidney injury with presence of neurological features.

Table 21: Influence of blood urea of day 7 on development of neurological features.

NEUROLOGICAL FEATURES	N	Y		Z	p-value
	Mean	Mean	SD		
UREA DAY 0	97.29	114.55	84.82	-1.574	0.117
UREA day 7	75.01	101.89	68.60	-2.756	0.006

Patients who had higher blood urea levels on day 7 were more likely to have neurological features on day 0 than patients with lower blood urea levels on day 7.

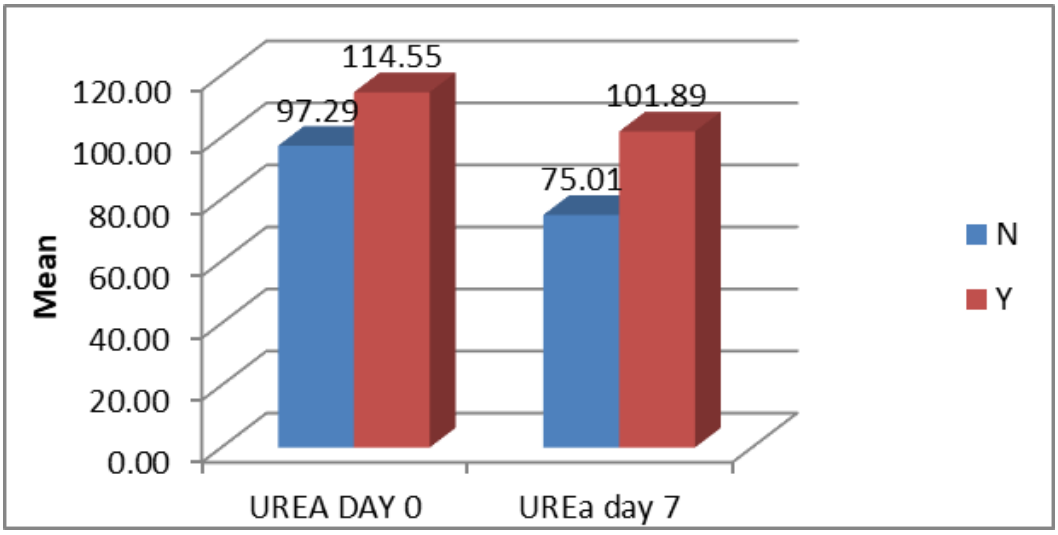


Figure 15: Influence of blood urea on day 7 with development of neurological features.

RENAL REPLACEMENT

Table 22: Patients with acute kidney injury requiring Renal replacement therapy.

Dialysis session	No. of cases	%
N	164	82.0%
Y	36	18.0%
Total	200	100.0%

In our study 36 patients which constituted 18 % of patients underwent renal replacement therapy.

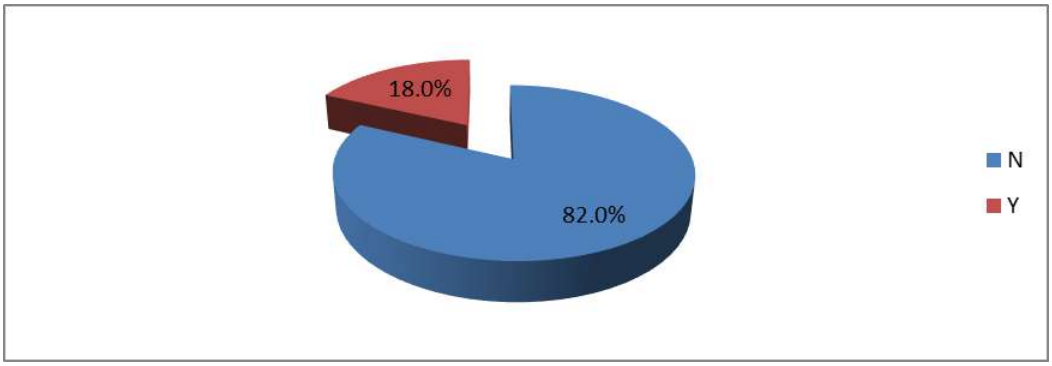


Figure 16: Patients with acute kidney injury requiring Renal replacement therapy.

Table 23 : Correlation of RRT and presence of neurological features.

		NEUROLOGICAL FEATURES				Total	Chi-square value	p-value
		NO NUMBER	NO %	YES NUMBER	YES %			
Dialysis session	No	135	86.5%	29	65.9%	164	9.895	0.002
	Yes	21	13.5%	15	34.1%	36		
Total		156	100.0%	44	100.0%	200		

Patients who underwent renal replacement were more likely to have had neurological complications with significant p value of 0.002.

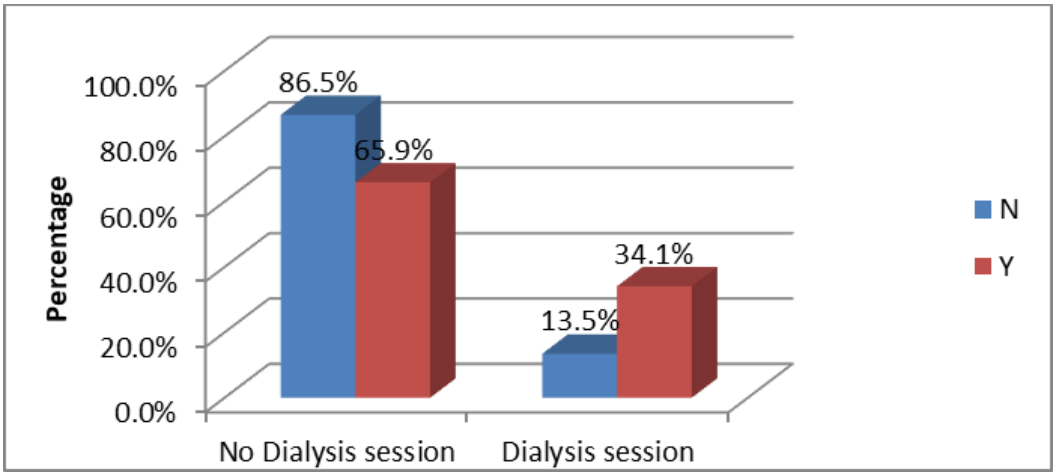


Figure 17: Correlation of RRT and presence of neurological features.

NEUROLOGICAL FEATURES

Table 24: Patients with acute kidney injury developing neurological features.

NEUROLOGICAL FEATURES yes/no	No. of cases	%
N	156	78.0%
Y	44	22.0%
Total	200	100.0%

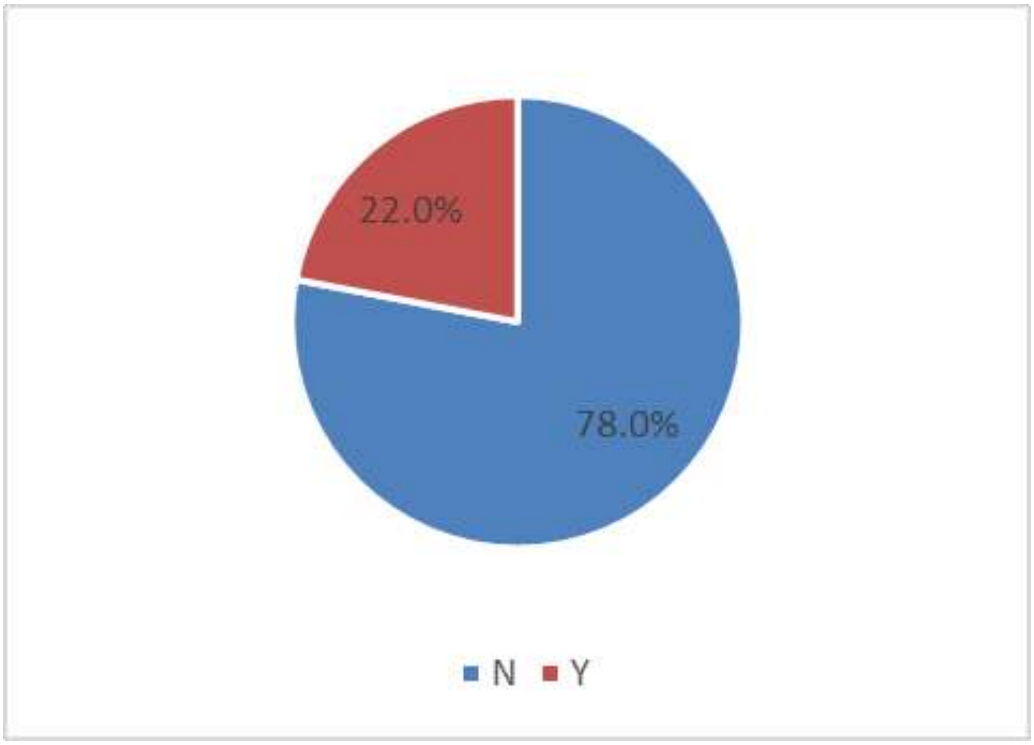


Figure 18 : Patients with acute kidney injury developing neurological features.

In our study neurological features were present in 44 patients out of total 200 patients which constituted 22 % of all patients

Table 25: CAM ICU score in patients with acute kidney injury.

	Day 0		Day 7	
CAM ICU DAY 0 (CONFUSION ASSESSMENT METHOD FOR ICU) POSITIVE FOR DELIRIUM	**No. of cases**	**%**	**No. of cases**	**%**
N	170	85.0%	181	94.8%
Y	30	15.0%	10	5.2%
Total	200	100.0%	191	100.0%

On day 0 Cam ICU score was positive in 30 patients which was 15 % of 200 patients. On day 7 CAM ICU score was positive in 10 patients. This was 5.2 % of the 191 patients present for follow up on day 7.

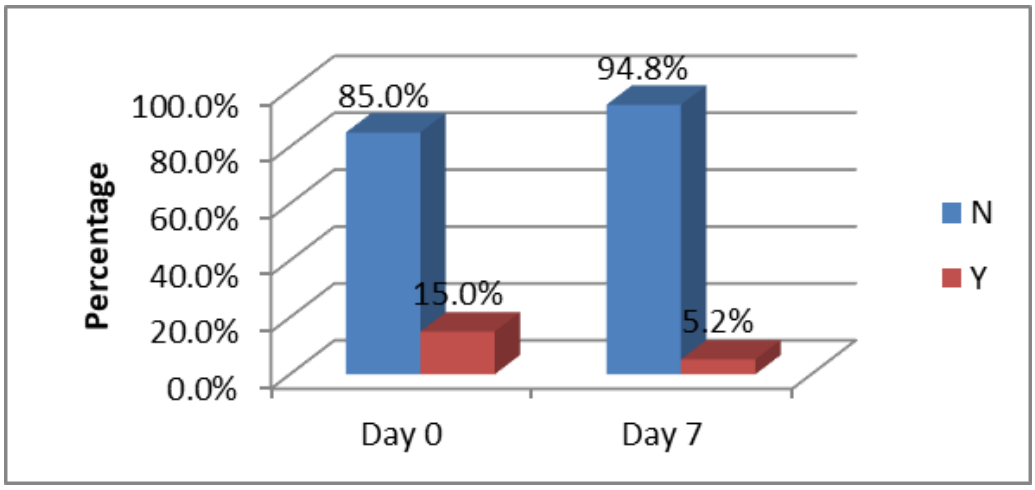


Figure 19: CAM ICU score in patients with acute kidney injury

MYOCLONUS

Table 26: Myoclonus in patients with acute kidney injury.

	Day 0		Day 7	
MYCOLONUS DAY 0	**No. of cases**	**%**	**No. of cases**	**%**
N	185	92.5%	191	100.0%
Y	15	7.5%	0	0.0%
Total	200	100.0%	191	100.0%

Myoclonus was present in 15 patients on day 0 which was 7.5 % of all patients and in no patient on day 7.

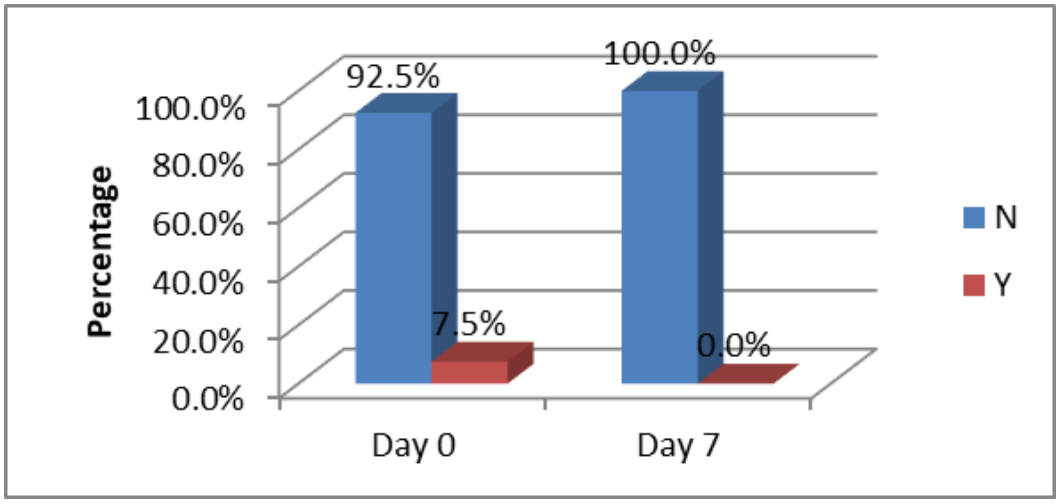


Figure 20: Myoclonus in patients with acute kidney injury

DELUSION/HALLUCINATION

Table 27: Delusions/Hallucinations in patients with Acute kidney injury.

	Day 0		Day 7	
DELUSION/HALLUCINATION DAY 0	**No. of cases**	**%**	**No. of cases**	**%**
N	187	93.5%	190	99.5%
Y	13	6.5%	1	0.5%
Total	200	100.0%	191	100.0%

Delusion or hallucinations were present in 13 patients which was 6.5% % of 200 patients on day 0 and in 1 patient on day 7.

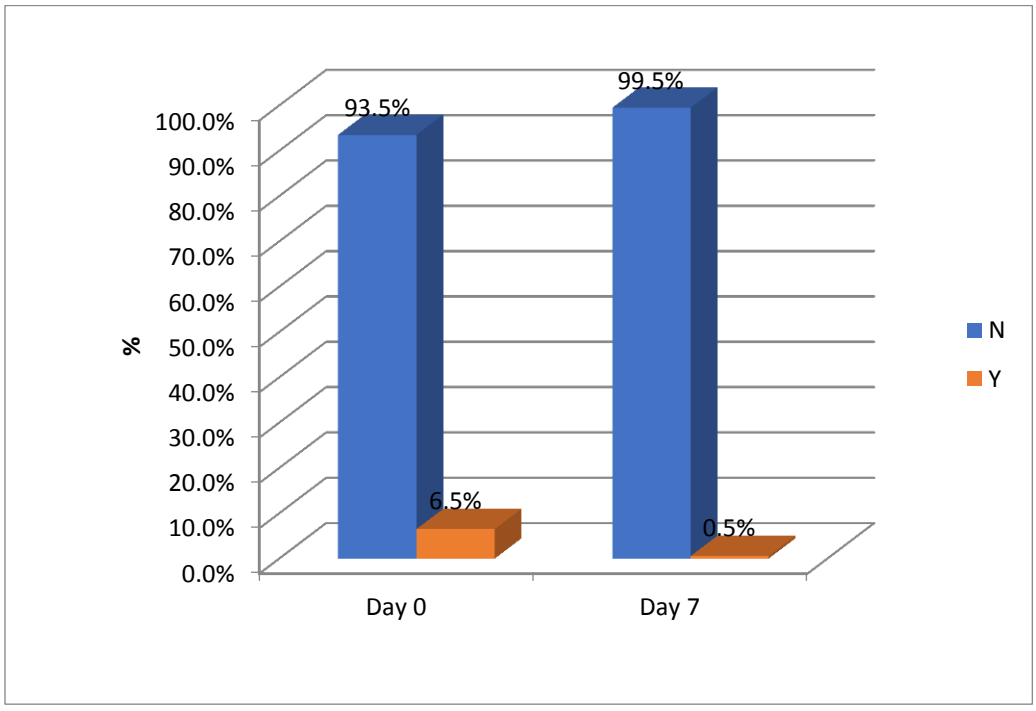


Figure 21: Delusions/Hallucinations in patients with Acute kidney injury.

ASTERIXIS

Table 28: Asterixis in patients with acute kidney injury.

	Day 0		Day 7	
Asterixis	**No. of cases**	**%**	**No. of cases**	**%**
N	191	95.5%	187	97.9%
Y	9	4.5%	4	2.1%
Total	200	100.0%	191	100.0%

Asterixis was present in 9 patients on day 0 which was 4.5 % of 200 patients and in 4 patients on day 7 which was 2.1 % of total 191 patients.

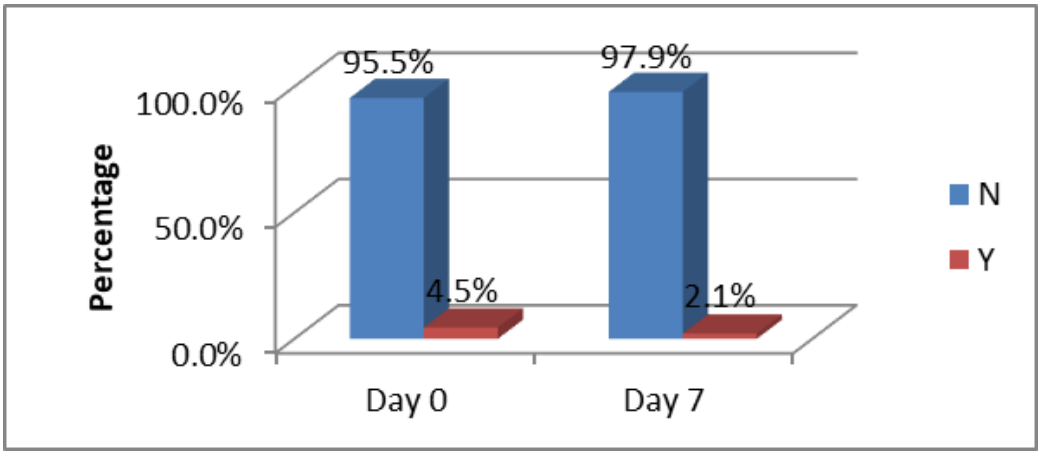


Figure 22: Asterixis in patients with acute kidney injury.

SEIZURES

Table 29:Seizures in patients with acute kidney injury.

	Day 0		Day 7	
Seizure	**No. of cases**	**%**	**No. of cases**	**%**
N	185	92.5%	190	99.5%
Y	15	7.5%	1	0.5%
Total	200	100.0%	191	100.0%

Seizures were present in 15 patients on day 0 which constituted 7.5 % of total patients and in 1 patient on day 7 which was 0.5 % of the patients available at follow up.

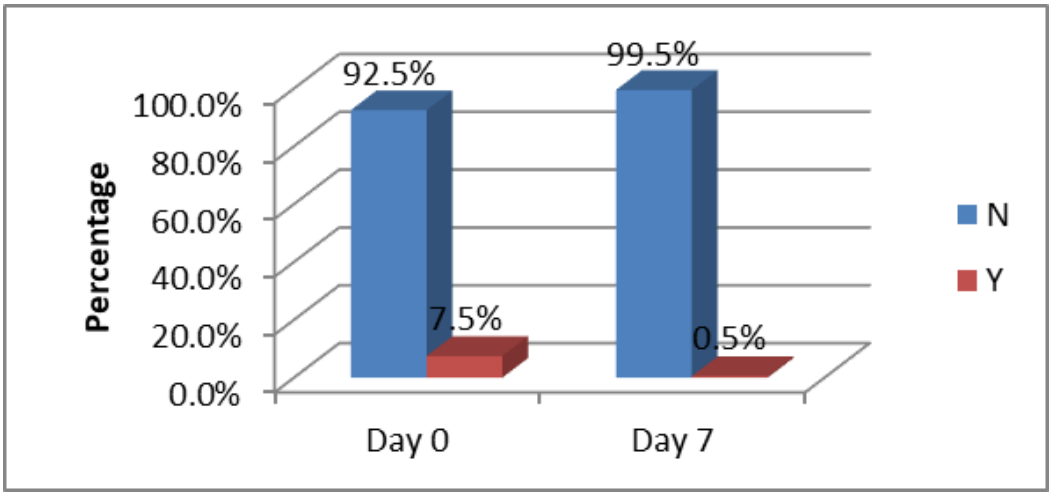


Figure 23: Seizures in patients with acute kidney injury.

SLEEP DISTURBANCE

Table 30: Sleep disturbances in patients with acute kidney injury.

	Day 0		Day 7	
Sleep disturbance	**No. of cases**	**%**	**No. of cases**	**%**
N	177	88.5%	179	93.7%
Y	23	11.5%	12	6.3%
Total	200	100.0%	191	100.0%

Sleep disturbance including sleep inversion, insomnia, excessive in somnolence were present in 23 patients on day 0 which was 11.5 % of 200 patients and on day 7 in 12 patients which was 6.3 % of the patients available for follow up.

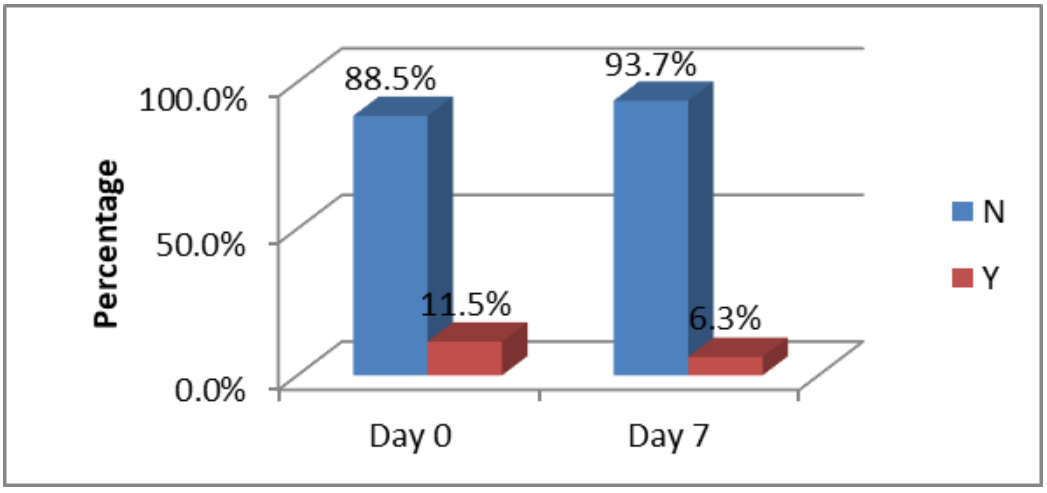


Figure 24: Sleep disturbances in patients with acute kidney injury

TREMORS

Table 31: Involuntary movements in patients with acute kidney injury.

	Day 0		Day 7	
Tremor/involuntary movements	**No. of cases**	**%**	**No. of cases**	**%**
N	187	93.5%	187	97.9%
Y	13	6.5%	4	2.1%
Total	200	100.0%	191	100.0%

13 patients had involuntary movements including tremors on day 0 which was 6.5 % of total 200 patients. 4 patients had involuntary movements on day 7 which was 2.1 % of the 191 patients present on follow up.

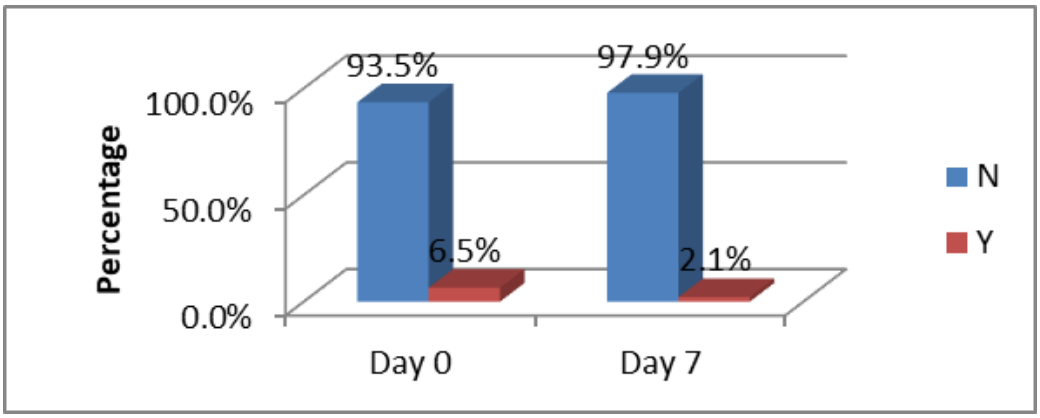


Figure 25: Involuntary movements in patients with acute kidney injury.

No patient had aphasia /cranial nerve involvement/ fasciculation or sensory abnormality that could be elicited on clinical examination on day 0 or day 7.

WASTING

Table 32: Wasting in patients with acute kidney injury.

	Day 0		Day 7	
Wasting	**No. of cases**	**%**	**No. of cases**	**%**
N	196	98.0%	187	97.9%
Y	4	2.0%	4	2.1%
Total	200	100.0%	191	100.0%

4 patients had wasting as determined by mid upper arm circumference less than 24 cm.

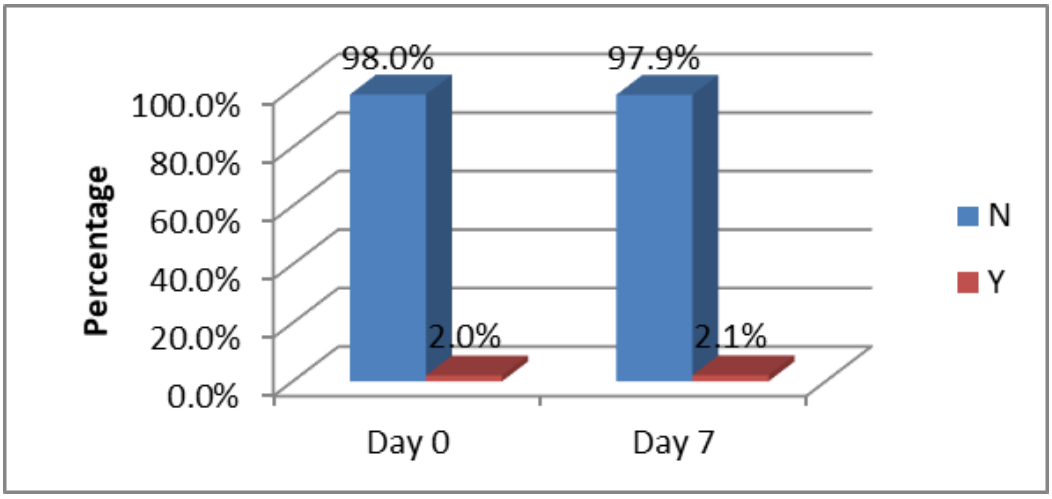


Figure 26 : Wasting in patients with acute kidney injury.

No patient had aphasia /cranial nerve involvement/ fasciculation /urinary incontinence/ night diarrhoea or sensory abnormality that could be elicited on clinical examination/history on day 0 or day 7.

NECK RIGIDITY

Table 33: Neck rigidity in patients with acute kidney injury.

	Day 0		Day 7	
Neck rigidity	**No. of cases**	**%**	**No. of cases**	**%**
N	192	96.0%	190	99.5%
Y	8	4.0%	1	0.5%
Total	200	100.0%	191	100.0%

8 patients had neck rigidity on day 0 which was 4 % of all patients and 1 patient had neck rigidity on day 7 which was 0.5 % of the 191 patients present at follow up.

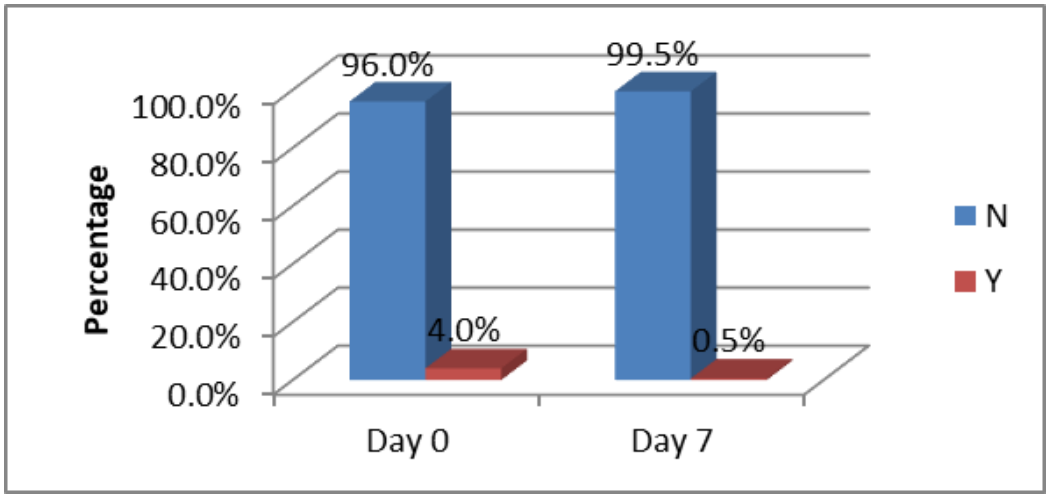


Figure 27: Neck rigidity in patients with acute kidney injury.

Table 34: Neurological spectrum in patients with acute kidney injury.

	Day 0		Day 7	
NEUROLOGICAL FEATURES	**No. of cases**	**% Of patient with neurological features**	**No. of cases**	**% Of patient with neurological features**
CAM ICU (DELIRIUM)	30	68.2%	10	22.7%
Myoclonus	15	34.1%	0	0.0%
Delusion/Hallucination	13	29.5%	1	2.3%
Asterixis	9	20.5%	4	9.1%
Seizure	15	34.1%	1	2.3%
Sleep Inversion	23	52.3%	12	27.3%
Tremor/Involuntary Movements	13	29.5%	4	9.1%
Wasting	4	9.1%	4	9.1%
Neck Rigidity	8	18.2%	1	2.3%

In our study the most common neurological presentation was of delirium which was present in 68.2 % of patients, followe-d by sleep inversion in 52.3 % of patients and seizures in 34.1 % patients

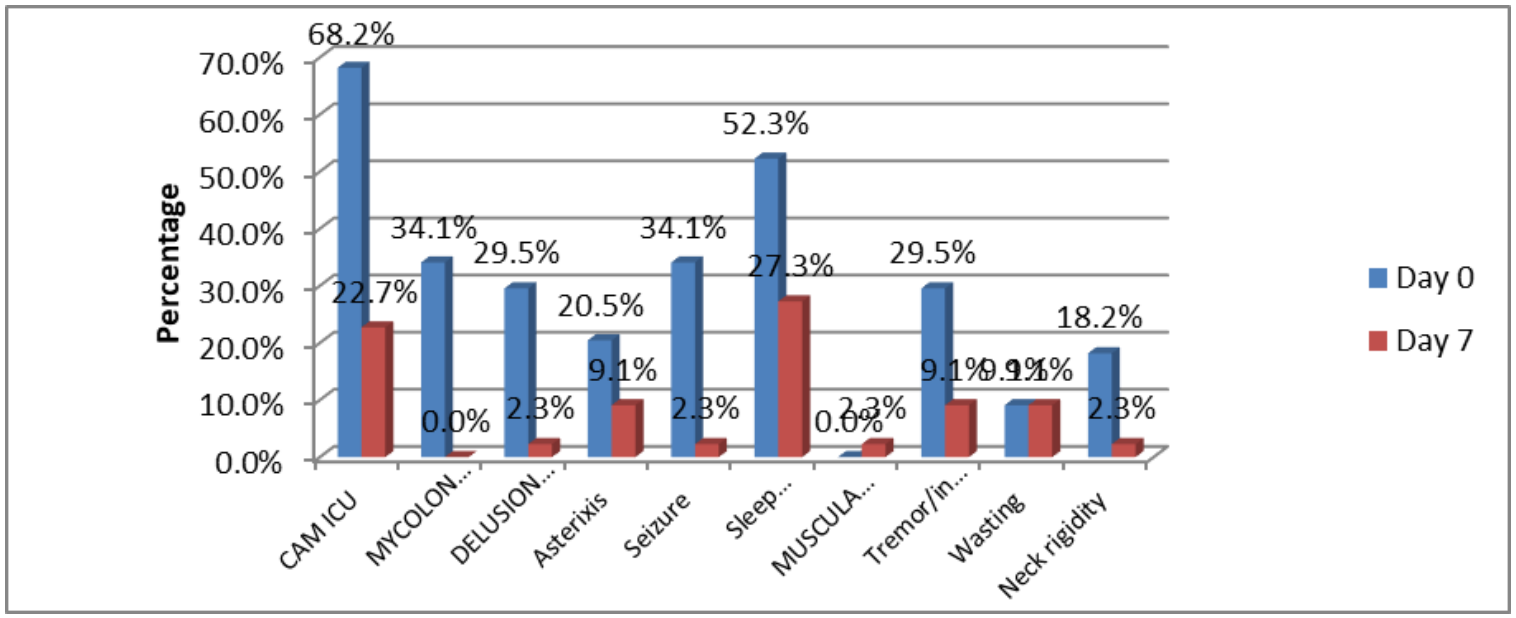


Figure 28: Neurological spectrum in patients with acute kidney injury.

RADIOLOGICAL INVESTIGATIONS

Table 35: CT head findings in patients with acute kidney injury.

CT HEAD	No. of cases	Percentage
CEREBRAL EDEMA	1	0.5%
HYDROCEPHALUS	1	0.5%
HYPODENSITIES B/L PERIVENTRICULAR WHITE MATTER	1	0.5%
NEUROCYSTICEROCIS	1	0.5%
NORMAL	16	8.0%
VENTRICULOMEGALY,ATROPHY	1	0.5%
NOT DONE	179	89.5%

CT head was done in 21 patients. It was normal in 16 patients. Abnormal findings in rest 6 patients included cerebral oedema; hypodensities bilateral periventricular white matter; hydrocephalus; neurocysticercosis; ventriculomegaly and atrophy present.

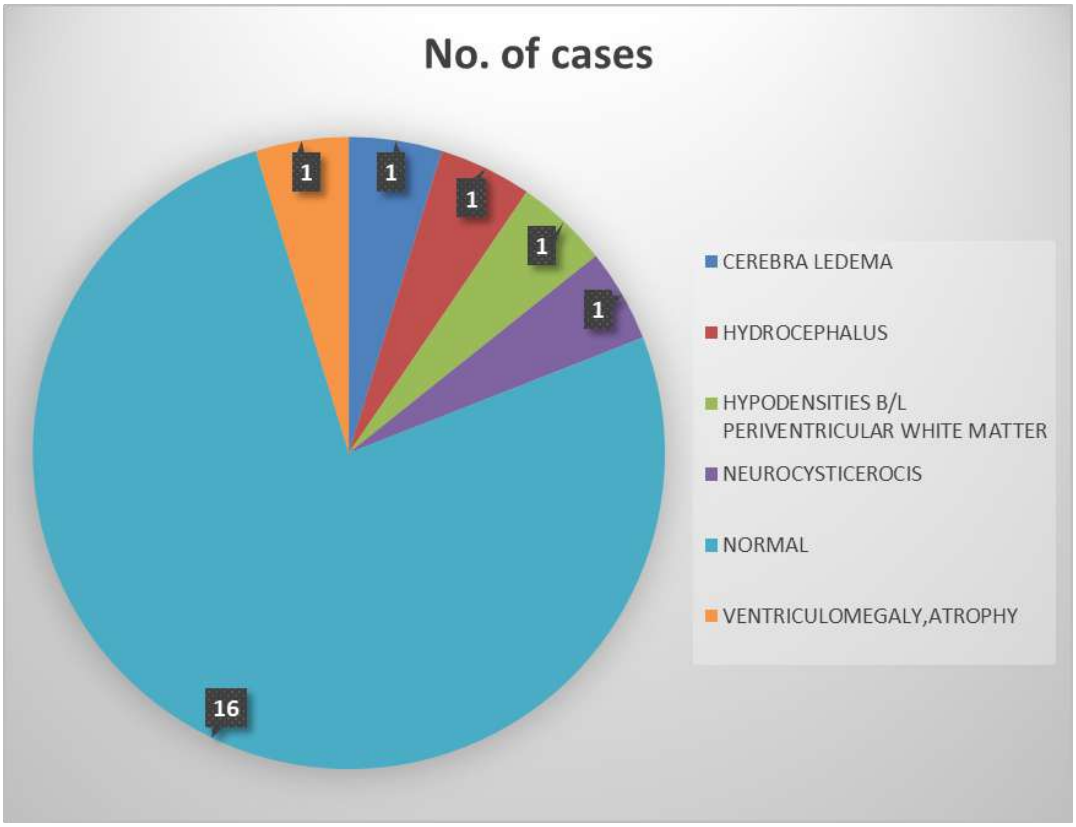


Figure 29: CT head findings in patients with acute kidney injury.

MRI BRAIN

Table 36: MRI Brain Findings In Patients With Acute Kidney Injury And Neurological Features

MRI	No. of cases	Percentage
DIFFUSE CEREBRAL EDEMA WITH B/L BASAL BASAL GANGLIA INVOLVED	1	0.50%
BILATERAL T2/FLAIR HYPERINTENSITY BILATERAL BASAL GANGLIA	1	0.50%
DIFFUSION EDEMA WITH DIFFUSION RESTRICTION BILATERAL CENTRUM SEMIOVALE	1	0.50%
POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME	1	0
ANGIOINVASIVE FUNGALINFECTIOM/MYCOTIC	1	0.5%
MULTIPLE EMBOLIC INFARCT	1	0.5%
FOCAL INCREASE T2OLIVARYNUCLEUS	1	0.5%
FRONTOTEMPORALDEMENTIA	1	0.5%
MASTOIDITIS,CHRONICATOPHY	1	0.5%
MILD SMOOTH DURAL THICKENING	1	0.5%
NORMAL	3	1.5%
PACHYMENINGITIS	1	0.5%
PACHYMENINGITIS,SUBDURALEMPYEMA,VENTRICULITIS,HYDROCEPHALUS	1	0.5%
NOT DONE	185	92.5%
Total	200	100.0%

MRI brain was done in 15 patients.3 patients had normal imaging.

I. One patient had diffuse cerebral edema with bilateral basal ganglia involvement.
II. One patient had T2/FLAIR hyper intensity in bilateral basal ganglia
III. One patient had diffuse edema with diffusion restriction in bilateral centrum semiovale.
IV. One patient showed imaging findings consistent with PRES as symmetrical T2/FLAIR hyperintensity of cortical and subcortical white matter in bilateral frontoparietal and occipital region.
V. One patient had multifocal acute infarcts, tiny foci of blooming in bilateral cerebellar and cerebral hemispheres with bilateral cerebellar hemispheres showing T2W/FLAIR hyperintense signal.
VI. One patient had mild smooth dural thickening along bilateral cerebral hemispheres suggestive of meningitis.
VII. One patient had meningeal enhancement and thickening.
VIII. One patient had focal increase in signal intensity on t2w in inferior olivary nucleus left sided? hypertrophic olivary degeneration.
IX. One patient had hyperintense foci on T2 W/FLAIR images without any diffusion restriction in bilateral periventricular and subcortical white matter and features of frontotemporal dementia.
X. One patient had left parietal region hyperintensity with blooming SW images, saccular outpouching enhancing parallel to vessel adjacent to this lesion suggestive of mycotic aneurysm. Multiple foci of blooming in bilateral cerebral and cerebellar hemispheres suggestive of microhaemorrhage. Multiple foci of calcification in bilateral cerebral hemispheres suggestive of granulomas. With findings suggestive of Angio invasive fungal infection.
XI. One patient had Pachymeningitis, subdural empyema, ventriculitis, hydrocephalus.
XII. One patient had mastoiditis, chronic cerebral atrophy

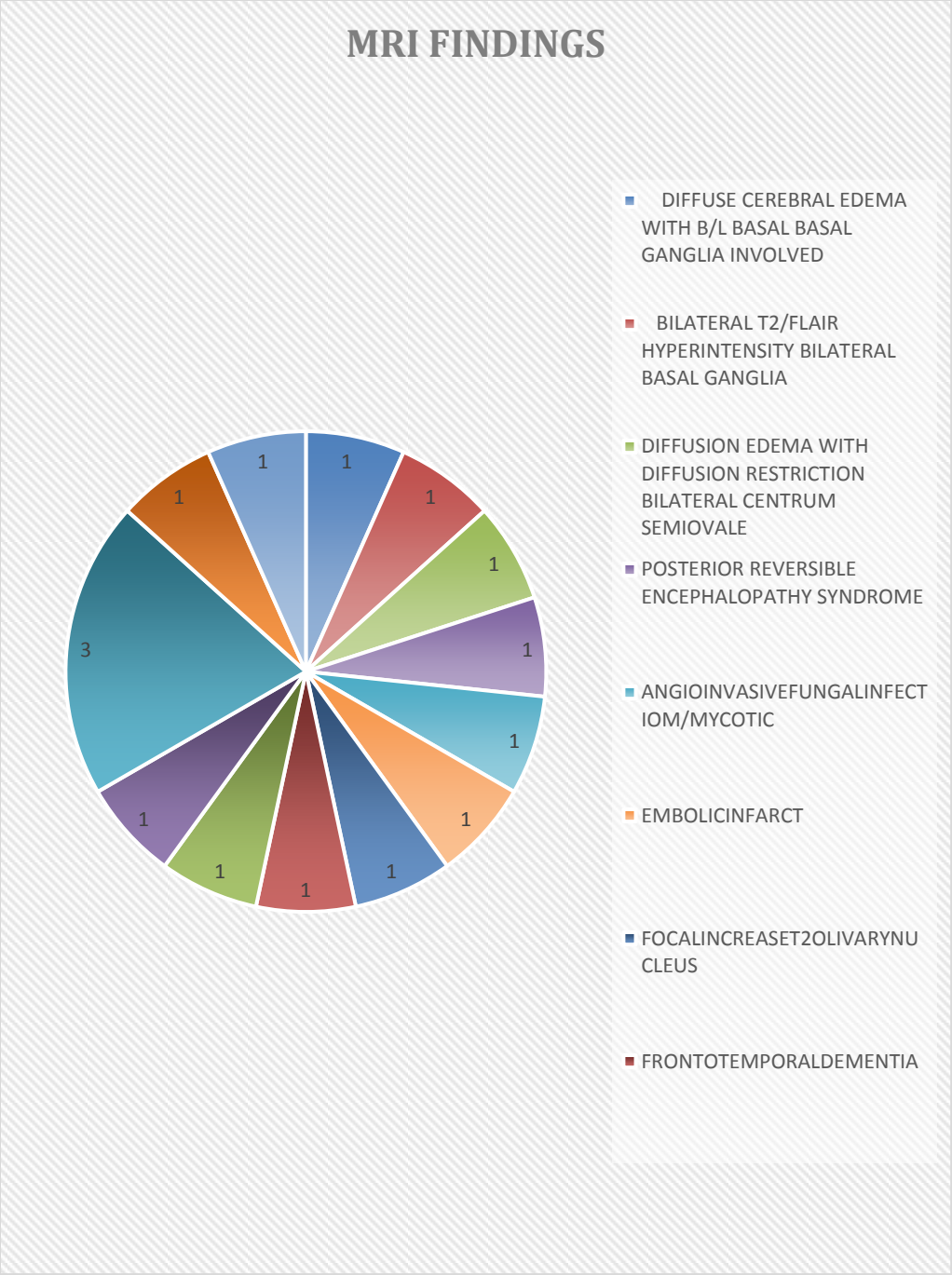


Figure 30:- MRI brain findings in patients with acute kidney injury.

EEG

EEG was done in 1 patient who had generalised slowing suggestive of diffuse encephalopathy.

CSF ANALYSIS

Table 37: Detailed CSF findings of patients with acute kidney injury.

	TLC	MNC	PML	Protein	Glucose	Cl	CSF
1	2	0	0	33	141	116	NORMAL
2	80	64	16	117	88	129	MONONUCLEAR CELL PREDOMINANT, HIGH PROTEIN
3	2	0	0	37	150	121	NORMAL
4	2	0	0	37	150	121	NORMAL
5	90	64	26	102	42	74	MONONUCLEAR CELL PREDOMINANT, HIGH PROTEIN, LOW GLUCOSE, HIGH ADA
6	2	0	0	28	91	118	NORMAL
7	2	0	0	37	40	126	NORMAL
8	3	0	0	98	78	102	HIGH PROTEIN
9	540	400	140	90	38	78	NEUTROPHILIC PREDOMINANT LOW GLUCOSE

CSF analysis was done in 9 patients. It was normal in 5 patients. 1 patient had fluid which was neutrophilic predominant and with low glucose; 1 patient had mononuclear cell predominant with high protein, low glucose, and high ADA; 1 patient had mononuclear cell predominant fluid with high proteins;1 patient had high protein acellular CSF analysis.

DISCUSSION

Neurological disturbances and acute kidney injury commonly coexist in patients. This is secondary to retention of uremic compounds, drug interactions and multiorgan disease process. These features have been well described in chronic kidney disease however there is lacunae for research about uremic encephalopathy in setting of acute kidney injury. A single centre study was conducted at Dayanand Medical college and hospital. 200 patients with acute kidney injury at presentation were included in the study. The peak incidence of patients with acute kidney injury was seen in 6th decade i.e. 51-60 years (34.5%). In study by Bien et al the incidence of acute kidney injury was maximum in >75 years age group.[83] Narinder pal et al found that the majority of patients belonged to age group of more than 65 years. Ravinder Mehta observed that in his study on patients with acute kidney injury the mean age was 59.5 years.[84] In an Indian study conducted by Eswarappa M et al, majority of patients with acute kidney injury belonged to the age group of 61-70 years[85] Anatomical and physiological changes that occur in the kidney with advancing age might be the reason for increased incidence of acute kidney injury among elderly patients. Other factors can be more comorbidities, increased severity of acute illnesses, polypharmacy, need for invasive procedures, and age-dependent changes in this population.

In our study, 58 % of patients were males and 42 % were females. This is like previous studies which have shown male preponderance. Ravindra L Mehta et al, in his study observed that 41% of patients were females and 59% were males.[84] Loutradis et al found that Incidence of acute kidney injury was higher in men compared to women (11.3% vs 7.1%; $P < 0.001$) after adjustment for age, eGFR, smoking, alcohol consumption, ethnicity and existing comorbidities. This is likely secondary to protective effect of oestrogens which reduce post-ischemic glomerular endothelial hyperpermeability in females.[86] [87]

In our study 68 patients (34%) had previous history of Hypertension, 68 patients (34%) had Type 2 Diabetes Mellitus , 32 patients (16%) had previously diagnosed CAD and 16 patients (8%) had previously diagnosed chronic liver disease. A study by Abebe et al observed that underlying comorbidities identified were Hypertension in 43% of patients , heart failure in 31% of patients and Type 2 Diabetes Mellitus in 14% of patients.[40] Patients with underlying Hypertension were more likely to have neurological complications than non-hypertensive patients with p value of 0.046. Hypertension itself can cause neurological complications. It has been estimated that approximately 1% of patients with Hypertension develop a hypertensive crisis during their lifetime.[88]

We had 42 patients (21%) with history of substance abuse including alcohol, opioid and other intravenous drugs. This was consistent with findings of Saxena et al where 34 patients (14.8%) were current smokers and 41 patients(17.9%) were alcohol drinkers.[89] In our study 16 patients (8%) had history of nephrotoxic drugs.

We observed that one third of the total patients (33.5%) required ICU monitoring. In a study by Minja et al 55 % of patients in ICU were diagnosed with acute kidney injury.[90] Multiple factors contribute to ICU stay for patients with acute kidney injury including possible need for RRT, ventilatory support and need for ionotropic support. In our study 9 patients (4.5 %) required ventilatory support. This was not in accordance with previous studies viz. Kulkarni et al with 26 % of patients and Saxena et al with 30 % of patients requiring ventilatory support.[89,91] The possible reason for this variation could be because these studies were done on patients admitted in only intensive care units. In our study it was observed that the patients who had to be admitted to ICU also had more incidence of neurological complications with significant p value of 0.001.This could be because patients with altered neurological status need more careful and vigilant monitoring hence justifying ICU stay in these patients. In our

study 40 patients (20%) required ionotropic support. This was consistent with findings observed by Singhal et al where 20.6 % of patients with acute kidney injury had hypotension.[92] In a study by Nagamani et al hypotension was present in 30 % of patients.[93] In a study by Kulkarni et al it was observed that 37 % of patients with AKI required inotropic support.[91] Hypotension is a cause for prerenal acute kidney injury and its presence in patients with acute kidney injury has been found to be associated with increased mortality. Presence of hypotension also influences the choice of renal replacement mode. In our study requirement of ionotropic support was associated with more likelihood of having neurological features with p value of <0.001. This is likely secondary to loss of cerebral autoregulation which has been associated with delirium.[94] Septic encephalopathy has also been found to occur more frequently in patients with lower MAP.[45] Our findings were similar to those observed by Jackal et al where mechanical ventilation and shock were independent predictors for development of delirium.[91]

It was observed that the most common cause of Acute kidney injury was infections which accounted for 117(58.5%) patients followed by tropical fever which accounted for 22(11%) patients and miscellaneous causes including hypercalcemia, ANCA vasculitis, HUS which together accounted for 22% of patients. These findings were similar to those seen by Narinder et al where infections were the most common cause of acute kidney injury.[95] In our study the most common source of infection was gastrointestinal(21.5%) followed by urosepsis(18.5%) and thirdly respiratory(10.5%). This was consistent with the findings of Nagamani et al where the most common infectious cause for AKI was gastroenteritis.[93] In our study obstructive uropathy accounted for 2 patients which was 1% of all patients. This was similar to contribution of obstructive uropathy to AKI seen in previous studies by Nagamani et al and Singhal AS et al. [92,93] We found that underlying sepsis was present in 27 (13.5%) patients as defined by qSOFA score more than or equal to 2 with presence of clinical

evidence of infection. Septic shock was present in 20 patients which accounted for 74 % of patients in sepsis. Neurological features were present in 48% of patients with sepsis and in 55 % of patients with septic shock. Hence significant correlation was found between patients who were in sepsis and septic shock with patients who had neurological complications with p value of 0.001. A study by Sprung et al observed altered mental status in 23% of patients with sepsis.[96]

In our study 36 patients(18%) underwent renal replacement therapy. Nagamani et al observed that of 50 cases, 36(72%) patients were managed conservatively and 14(28%) patients underwent haemodialysis.[93] We found significant correlation between patients who underwent renal replacement and those who developed neurological complications with p value of 0.002. This is likely secondary to two factors, firstly uremic encephalopathy itself is a common indication for initiation of renal replacement.[97] Secondly, dialysis itself has been found to be associated with range of complications including Cerebral haemorrhage, Cerebral thrombosis, Central pontine myelinolysis, Wernicke's encephalopathy, Dialysis dementia, Cognitive impairment, Disequilibrium syndrome, Anterior ischaemic optic neuropathy, and Posterior reversible encephalopathy syndrome. [17] One patient in our study presented with delirium and seizures after a session of haemodialysis done outside one day prior to admission. Possibilities of dialysis disequilibrium and sepsis associated encephalopathy were kept. [98] [99] Previous studies have also observed cognitive deficit in patients undergoing haemodialysis. Murray M enrolled 338 haemodialysis subjects and performed detailed 45-minute neuropsychological battery and observed 37% of subjects had severe, 36% had moderate, and 14% had mild cognitive impairment; only 13% had normal cognitive function. Haemodialysis patients were more than three times as likely to have severe cognitive impairment than a comparison group of non-CKD patients.[100] However, it has not been possible so far to differentiate how much of the cognitive impairment

observed in haemodialysis patients is pre-existing, due to uraemia or due to the dialysis process itself. All the clinical studies done so far have studied the dialysis related neurological complications in patients with end stage renal disease who are more predisposed to these complications.

Urine culture was sent in 95 patients. Specific organism was isolated in 15 patients. Most common organism was Escherichia Coli(n=6) followed by Klebsiella (n=3). Other organisms seen were Candida stelloides ,Enterobacter Enterococcus and Serratia. Blood cultures were sent in 94 patients. 5 patients had a specific organism isolated which included Escherichia coli, Candida Giller, Klebsiella, Staphylococcus Hemolyticus and MRSA. Reasons for lack of isolation of organism in other patients include initiation of antibiotics prior to admission, difficult to grow organisms and occult source of infection. In our study patients with Enterococcus, Escherichia coli , Candida , Enterobacter isolated in urine , Staphylococcus hemolyticus isolated in blood had altered sensorium. Commonly implicated organisms predicting risk of encephalopathy include Enterococcus feacium, Acinetobacter spp., Pseudomonas aerugin osa, Staphylococcus aureus and Stenotrophomonas maltophilia.[46] It has been observed that patients with multiple bacteria on blood culture and with *Candida albicans* have more severe brain dysfunction and a higher mortality rate.[101]

Using the KDIGO criteria, it was seen that half of the patients(51%) belonged to Stage 1 acute kidney injury, 19.5 % of patients belonged to stage 2 Acute kidney injury and 29.5 % of patients belonged to stage 3 acute kidney injury. In a study by Abede et al it was observed 22.5% of patients were in stage 1, 45.5% were in stage 2 and 32% were in stage 3 of acute kidney injury.[102] In a study done by Kulkarni et al 22 % patients were in Stage 1, 44% were in Stage 2 and the remaining 34% were in Stage 3.[91] This discordance seen in distribution of patients is probably because these studies were done exclusively in ICU setting whereas we

included all the patients admitted via emergency. We did not find any correlation between the stages of acute kidney injury and likelihood of having neurological complications. We also did not find any correlation of blood urea or creatinine levels on day 0 with presence of neurological features. This is secondary to multiple mechanisms responsible for neurological complications like disrupted blood brain barrier integrity, inflammatory cascade, accumulation of uremic toxins, interaction of drugs and alteration in their pharmacokinetics and pharmacodynamics. More than 90 uremic toxins have been identified so far. Hence a single biomarker is not sufficient to predict neurological complications. In vitro studies have demonstrated direct effect on neurons of other uremic compounds which are more toxic than urea. These include uric acid, indoxy sulphate and methyl guanidine.[103] Our findings were consistent to those found in a cross sectional studies on 209 CKD patients where urea levels did not correlate with depression.[104] Similarly asterixis has also been found to poorly correlate to creatinine clearance and blood urea nitrogen (BUN) values.[105] However, higher blood urea levels on day 7 had correlation with development of neurological features with p value of 0.006. There could be a possibility that persistent uraemia if left untreated leads to neurological abnormalities.

In our study, out of 200 patients, 44 patients (22%) had neurological features. The most common clinical presentation was of delirium, characterized by altered consciousness with a reduced ability to focus, sustain, or shift attention that develops quickly and fluctuates over the course of the day.[106] CAM ICU score was used to screen for delirium and was positive in 30 patients which was 15% of total patients. On day 7 CAM ICU score was positive in 10 patients(5.2%). In a prospective study of 1487 patients, Zipser et al. found that acute kidney injury conferred a tenfold increased risk of delirium (OR 10.01, CI 1.13–88.73, $p = 0.039$) [107] while the BRAIN-ICU study found that acute kidney injury was present in 50% of study days when patients were delirious.[108] In a study by Jackel et

al delirium was detected in 60.1% of patients with acute kidney injury with independent predictors as age, psychiatric disease, alcohol abuse, mechanical ventilation, severe shock, and acute kidney injury stage.[109] A study by Pisani et al demonstrated that serum creatinine greater than 2 mg/dl, was associated with delirium during critical illness.[110] However, these studies did not exclude patients with underlying chronic kidney disease.

We found myoclonus to be present in 15 patients(7.5%) on day 0 and in no patient on day 7. In a study by Tyler et al 40 % of patients with renal failure had asterixis or marked fasciculations often with myoclonic phenomena.[111] Action myoclonus, stimulus-sensitive myoclonus and twitch-convulsive syndrome have been described in uremic encephalopathy.[98] No previous studies have been done to determine the incidence of myoclonus in patients with acute kidney injury although literature has stated it as a manifestation of uremic encephalopathy and septic encephalopathy.[112] [113]

Asterixis was present in 9 patients(4.5%) on day 0 and in 4 patients 2.1%) on day 7. It has been described secondary to many metabolic encephalopathies including uraemia, malnutrition, severe pulmonary disease, and polycythaemia rubra vera.[114] Case report has described presence of asterixis in an elderly female with hyper magnesia and chronic kidney disease.[115] However no study has evaluated incidence of asterixis in acute kidney injury.

We found sleep disturbances including sleep inversion, insomnia, excessive in somnolence were present in 23 patients(11.5%) on day 0 and in 12 patients (6%)on day 7. Previous studies have reported sleep abnormalities in patients with CKD. A study that included 1,643 patients from 335 US dialysis centres reported that 50% patients had trouble in falling asleep, 59% patients woke during the night, and 49% patients woke early in the morning; 53% reported one or more of these symptoms

at all or most of the time.[116] Multiple hypotheses have been proposed to account for uraemia-induced daytime sleepiness. These include subclinical uremic encephalopathy, abnormal metabolism and retention of melatonin, tyrosine deficiency leading to diminished neurotransmitters associated with arousal, alteration in body temperature rhythm resulting in a perturbation of the sleep-wakefulness cycle, release of sleep-inducing inflammatory cytokines during dialysis, the effects of dialysate temperature on sleep, and co-existent sleep apnea syndrome (SAS).[40] However, all these studies have been done in patients with end stage renal disease. No data is available for sleep disturbances in acute kidney injury.

In our study delusion or hallucinations were present in 13 patients(6.5%) on day 0. Mehta M et al described a case report of patient with acute kidney injury who presented with hallucinatory behaviour, delusions of persecution & reference and thought abnormalities including tangentiality & loosening of association.[117] Kelsey et al described a young male who presented with anxiety, agitation, delusion of persecution and acute kidney injury after excessive intake of energy drinks.[118] Stenbich & Haapanen found psychiatric disturbances in 58% of patients with chronic kidney disease which included clouding of consciousness, disorientation, delusions and hallucinations.[119] Burn and bates has describe presence of visual hallucinations, disorientation, and agitation in the late stages of uremic encephalopathy.[19] Brouns and De Dyns described auditory hallucination in dialysis encephalopathy.[17] No study has been done to estimate incidence of delirium or hallucinations in acute kidney injury.

Seizures were present in 15 patients(7.5%) on day 0 and in 1 patient(0.5%)on day 7. In a study by Hamed et al 15.6% of patients with CKD and uremic syndrome had recurrent seizures.[120] In general the incidence of uremic seizures has been described as 10%.[121] Development of seizures in renal disease can be secondary to aetiologies seen in the general population such as intracranial bleeding, Electrolyte

imbalance, PRES, Hypertensive urgency, and hypertensive emergency, tumours, infections, and intoxications or due to kidney-related aetiologies like Dialysis disequilibrium and Uremic encephalopathy. In a study by Nigro et al seizures were present in 5% of patients with hyponatremia. [122] Seizures in uremic encephalopathy likely occur secondary to increased guanidosuccinic acid, methyl guanidine in the cerebrospinal fluid.[123]

13 of our patients(6.5%) had tremors on day 0 and 4 patients(2.1%) had tremors on day 7. Data is not available for incidence of tremors in acute kidney injury however they have been described in AKI secondary to electrolyte imbalance(hyponatremia, hypernatremia, hypomagnesemia), uremic striatopalldial syndrome, dialysis disequilibrium, osmotic demyelination syndrome and dialysis dementia. Lithium toxicity manifests as action tremor, cerebellar ataxia, and renal involvement in the form of nephrogenic Diabetes insipidus and reduction in glomerular filtration rate.[124] Several other drugs (acyclovir, cephalosporin, etc.) may cause tremor, myoclonus, chorea, and ataxia in the setting of renal dysfunction.[125]

In our study 8 patients(4%) had neck rigidity on day 0 and 1 patient(0.5%) on day 7. Neck rigidity is a symptom of meningeal irritation secondary to infectious causes like meningitis (bacterial, viral, fungal) and non-infectious causes like drugs, subarachnoid haemorrhage, neurosarcoidosis and systemic lupus erythematosus. No previous study has been done to determine incidence of neck rigidity in AKI.

The percentage of patients with wasting as determined by measuring mid upper arm circumference was found to be 2%. We used the cut-off of less than 24 cm to determine wasting as proposed by Tang et al.[126] In a study by Berbel et al prevalence of protein energy wasting in acute kidney injury as determined by SGA (Subjective Global Assessment), biochemical parameters, anthropometry, and bioelectrical impedance analysis (BIA) was found to be 66.7 %. [127] The low incidence of wasting observed in

our study could be because mid arm circumference alone is not a sensitive criteria to identify protein energy wasting in acute settings.[128]

In our study CT head was done in 21 patients. It was normal in 16 patients. The findings in rest of the 5 patients included cerebral oedema; hypodensities bilateral periventricular white matter; hydrocephalus; neurocysticercosis; ventriculomegaly and atrophy. We performed MRI Brain on 15 patients. 3 patients had normal imaging. The abnormal findings in rest of the 12 patients include cerebral edema,T2/FLAIR hyper intensity in bilateral basal ganglia, Toxic/metabolic encephalopathy, PRES, Multifocal acute infarcts with bilateral cerebellar edema, Meningitis (n=3), Hypertrophic olivary degeneration, Frontotemporal dementia, Angio invasive fungal infection, Mastoiditis and Chronic cerebral atrophy.

Previous studies have concluded that Imaging findings of Uremic encephalopathy can be classified into three types: Cortical or subcortical involvement, [129] Basal ganglia involvement causing both cytotoxic and vasogenic oedema[130] and White matter involvement. Asian population has been found to present only with only basal ganglia involvement.[131] In our study we had a patient with findings suggestive of PRES. Iwafuchi reported a 28 year old male who presented with uraemia without Hypertension with MRI brain showing increased signal intensities in the brainstem (pons and mid-brain) and bilateral (right > left) frontal centrum semiovale.[132] Similarly Khanal S et al reported a 28 year old female who presented with stage 3 AKI and had vasogenic oedema suggestive of PRES.[133] T2/FLAIR hyper intensity were found in bilateral basal ganglia of a patient. This finding was similar to that reported by Wen Yu Gong et al where T2FLAIR/MRI of a 32-year-old female on haemodialysis with involuntary movements showed symmetrical hyperintense non-haemorrhagic lesions in the entire bilateral basal ganglia.[134] Similar findings were found by Eun Ja Lee et al on neuroimaging of 4 patients with long-term Diabetes mellitus and severe azotaemia. [135]

In our study CSF analysis was done in 9 patients. Of these 9 patients, 1 patient had neutrophilic predominant fluid with low blood glucose; 1 patient had mononuclear cell predominant with low blood glucose and high ADA levels suggestive of Tuberculous meningitis.1 patient who was also PLHIV had mononuclear cell predominant high protein CSF fluid. 5 patients had CSF fluid analysis which was in normal range. Lastly 1 patient had high CSF fluid protein. Film array was not done in any of these patients. In this context there is possibility of uraemia having some contribution to the encephalopathy of the last 6 patients. Analysis of CSF in the uremic patient with meningism has been described to include presentation as aseptic meningitis, with up to 250 lymphocytes and polymorphonuclear leucocytes/mm^3. The CSF protein may also be increased up to 1.0 g/l as described by Burns and Bates.[19] Hence the presence of uraemia as a contributing factor cannot be ignored.

Hence, a wide spectrum of neurological features is seen in acute kidney injury. These complications have significant correlation with the hemodynamic profile , presence of sepsis and septic shock. Clinical and radiological findings have more important role than laboratory parameters in screening and diagnosing the patients with uremic encephalopathy.

STRENGTHS AND LIMITATIONS

STRENGTHS

To the best of our knowledge, it is the first study in India to study neurological spectrum in acute kidney injury exclusive of underlying chronic kidney disease. Neurological features in chronic kidney disease have been well described however similar studies have not been done in AKI. To the best of our knowledge, it is also the first study in India to describe the neurological spectrum of AKI with frequency of each neurological abnormality.

LIMITATIONS

We acknowledge limitations in present study. Neuroimaging could not be done in many patients because of lack of feasibility for shifting to radiology department . Few patients had co-existent meningitis, sepsis and electrolyte abnormalities which could also have contributed to altered sensorium. N-Terminal propeptide of CNP (NT-proCNP), protein S100b, and neuron-specific enolase (NSE) are biomarkers for sepsis associated encephalopathy which are not available at our centre, hence differentiation between uremic encephalopathy and SAE cannot be made reliably. Newer biomarkers are now being recommended for risk stratification of acute kidney injury which are not available as of now.

SUMMARY AND CONCLUSION

Acute kidney injury is associated with morbidity, mortality and long-term adverse consequences. Neurological complications can occur because of acute kidney injury, or both can be secondary to multi organ dysfunction.

- 200 patients admitted with acute kidney injury were studied.
- Majority of patients were male (58%) and had age more than 50 years.
- The most common cause of acute kidney injury was infections (59%).
- 44 patients (22%) had neurological complications.
- The most common neurological presentation in acute kidney injury was delirium in 68 % followed by sleep inversion in 52% and seizures in 34%.
- Hypertension and Type 2 Diabetes Mellitus were the most common underlying comorbidities however, only Hypertension was a risk factor for development of neurological complications.
- There was no correlation of staging of AKI with presence of neurological features.
- Presence of sepsis and septic shock were independent predictors for development of neurological complications in AKI along with higher qSOFA score, need for inotropes, need for ventilatory support and need for Renal replacement therapy.

  In conclusion our study demonstrates that patients with acute kidney injury can have neurological complications which occur secondary to a coexistent disease process or to only AKI or with contribution from both. Biochemistry and lab investigations at presentation are not sufficient to predict the development of neurological abnormalities. Hence physical examination and monitoring of hemodynamic parameters along with radiological findings assumes more important role.

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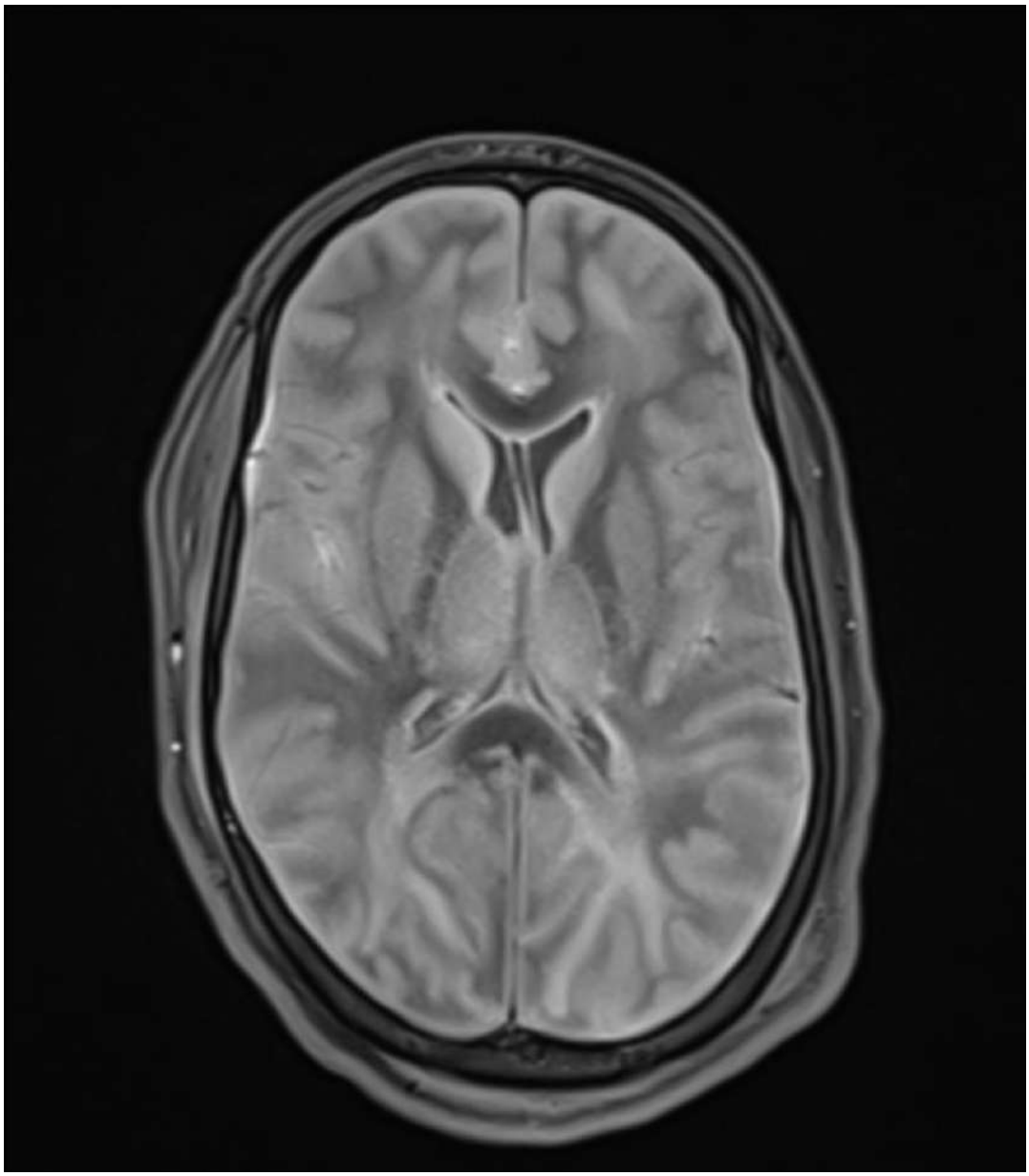
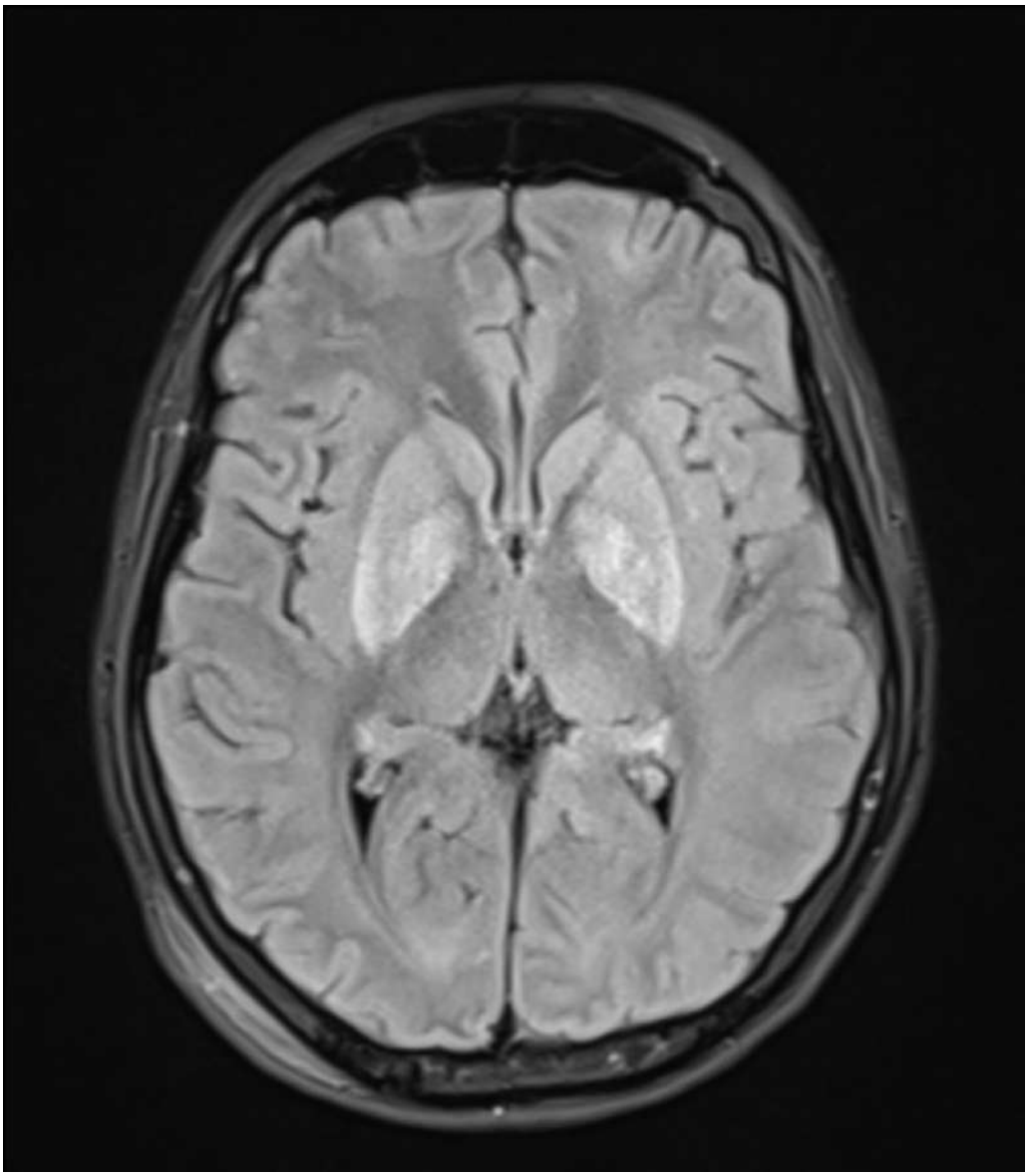
PICTURE 1: DIFFUSE CEREBRAL EDEMA



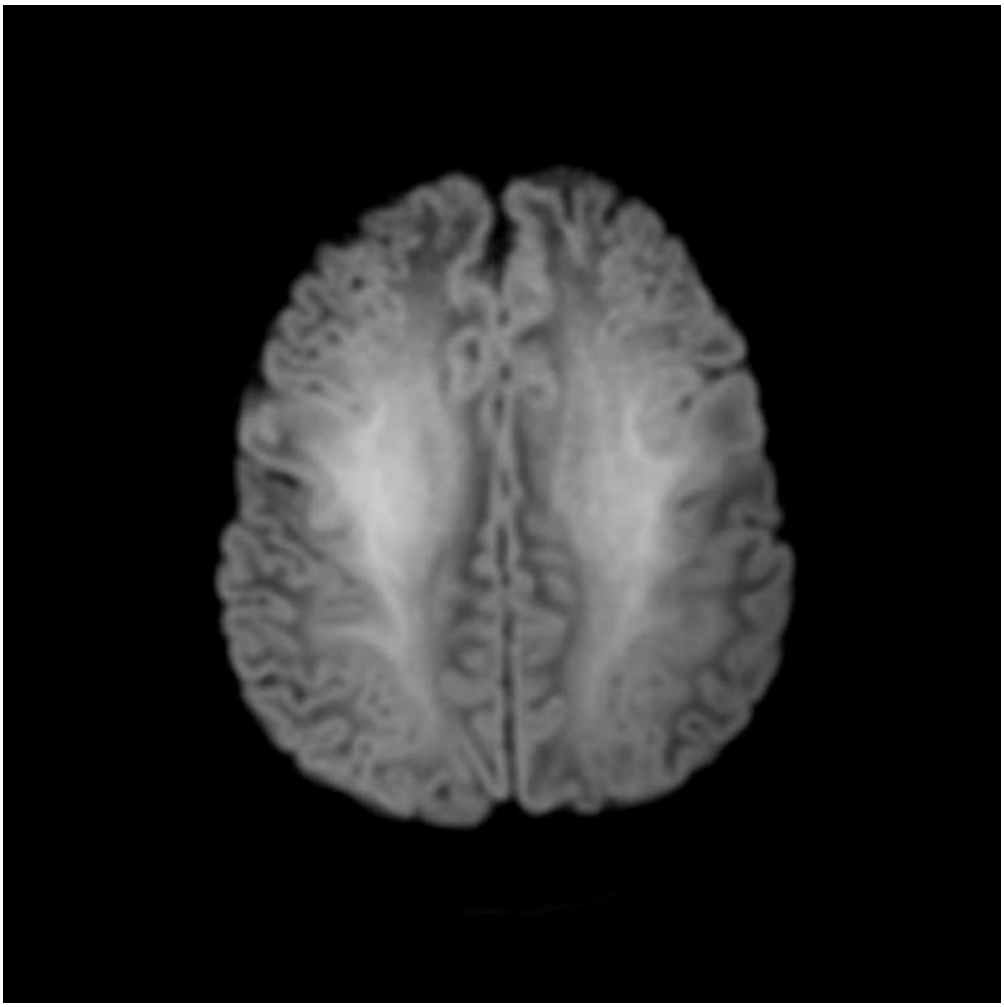
Axial Flair image showing diffuse oedema in bilateral cerebral hemispheres and hyperintensity in bilateral basal ganglia and thalamus.

PICTURE 2 : BASAL GANGLIA HYPERINTENSITY.



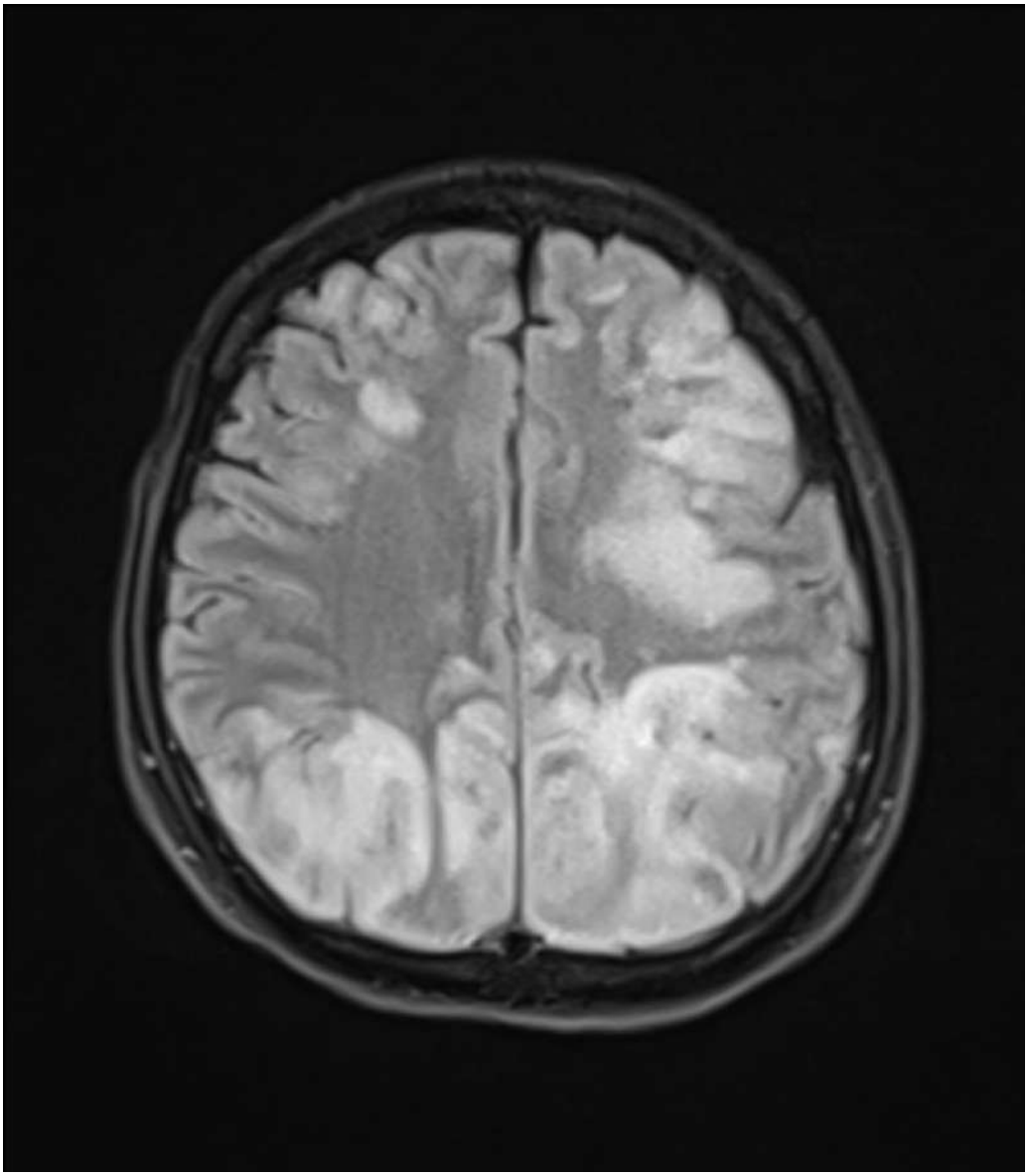
Axial flair image showing nearly symmetrical hyperintensity in bilateral globus pallidus and dorsal portion of bilateral putamina.

PICTURE 3: DIFFUSE CYTOTOXIC EDEMA



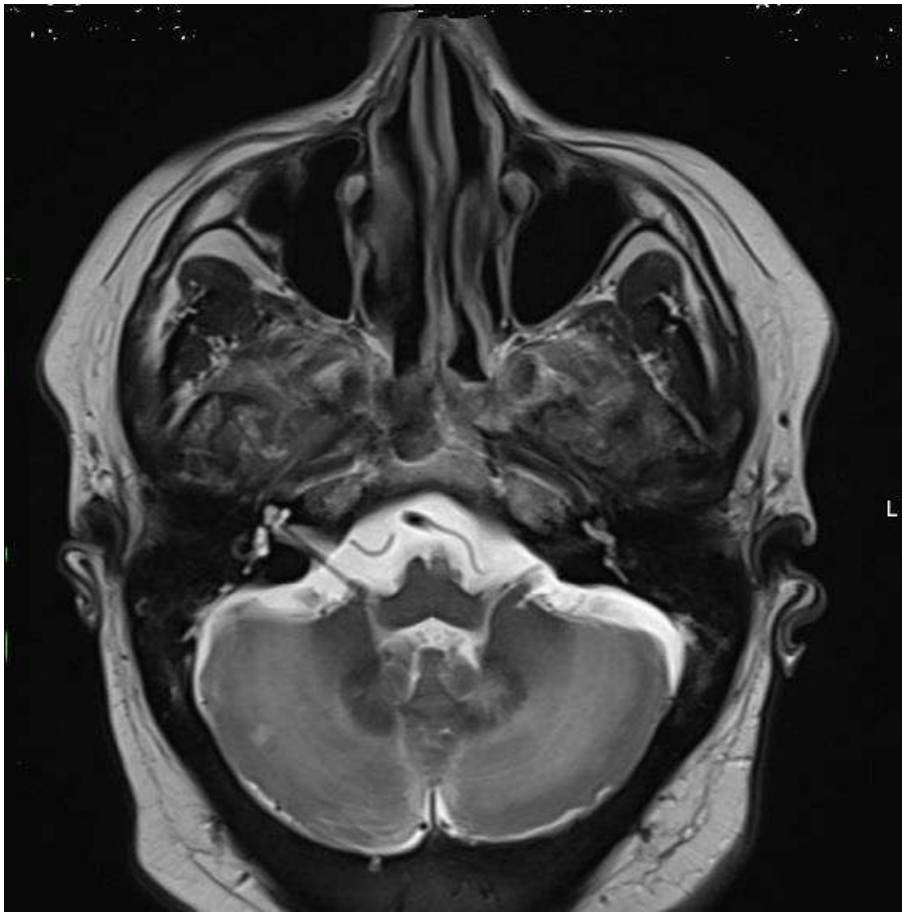
Axial diffusion weighted image showing cytotoxic oedema in bilateral semiovale.

PICTURE 4 : POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME



Axial t2 weighted image showing T2 hyperintensity involving cortex and subcortical white matter in bilateral frontoparietal region consistent with PRES.

PICTURE 5 : BILATERAL CEREBELLAR EDEMA



Axial T2 weighted image showing nearly symmetrical T2 hyperintensity in bilateral cerebellar hemispheres

PROFORMA

Name

Age

Sex

Presenting complaints-

ICU Stay-YES/NO

Comorbidities-hypertension

-diabetes mellitus 2

- CAD

Any nephrotoxic drugs prior to admission--YES/NO

Any neurotoxic drugs prior to admission-YES/NO

H/O Alcohol/smoking/substance abuse-YES/NO

Family history of neurological disease-YES/NO

Family history of kidney disease-YES/NO

Mechanical ventilatory support--YES/NO

Ionotropic support —YES/NO

qSOFA

	Day 0	Day 7
qSOFA		

Table 5 Sequential Organ Failure Assessment (Quick) qSOFA

Assessment	qSOFA score
Low blood pressure (SBP ≤100 mmHg)	1
High respiratory rate (≥22 breaths/min)	1
Altered mentation (GCS ≤14)	1

Signature of Candidate

Signature of Supervisor

AKI DIAGNOSIS

Stage	Serum creatinine	Urine output
1	1.5-1.9 times baseline OR ≥0.3 mg/dl (≥26.5 µmol/l) increase	<0.5 ml/kg/h for 6-12 h
2	2.0–2.9 times baseline	<0.5 ml/kg/h for ≥12 h
3	3.0 times baseline OR Increase in serum creatinine to ≥4.0 mg/dl (353.6 µmol/l) OR Initiation of renal replacement therapy OR, in patients <18 years, decrease in eGFR to <35 ml/min per 1.73 m^2	<0.3 ml/kg/h for ≥24 h OR Anuria for ≥12 h

Urine output **Blood urea**

Serum creatinine –

AKI STAGE-

Dialysis session-

	DAY 0	DAY 7
VITALS		
Blood pressure-		
Heart rate		
Respiratory rate		
Saturation oxygen		
	DAY 0	**DAY 7**
Lab values		
TLC		
DLC		
Hb		
Platelet count		
Sodium		
Potassium		
Chloride		
AST		
ALT		
ALP		
Bilirubin		
Total protein		
Albumin		
Calcium		

Signature of Candidate **Signature of Supervisor**

Magnesium

Phosphorus

Parathyroid (if done)

Urine R/E

UPCr(IF DONE)

USG abdomen-

ABG

	DAY O	DAY 7
pH		
pco2		
pO2		
Lactate		
Hco3		
So2		

Urine culture

Blood culture

Any other culture

NEUROLOGICAL FEATURES

Signature of Candidate

Signature of Supervisor

GLASGOW COMA SCALE	
Eye-opening (E)	
• Spontaneous	4
• To speech	3
• To pain	2
• Nil	1
Best motor response (M)	
• Obeys	6
• Localises	5
• Withdraws	4
• Abnormal flexion	3
• Extensor response	2
• Nil	1
Verbal response (V)	
• Orientated	5
• Confused conversation	4
• Inappropriate words	3
• Incomprehensible sounds	2
• Nil	1
Coma score = E + M + V	
• Minimum	3
• Maximum	15

	Day 0	Day 7
GCS-		

Scenario 1- pt is sedated

Score	Classification	(RASS)
+4	Combative	Overtly combative or violent; immediate danger to staff
+3	Very agitated	Pulls on or removes tube(s) or catheter(s) or has aggressive behavior toward staff
+2	Agitated	Frequent non-purposeful movement or patient–ventilator dyssynchrony
+1	Restless	Anxious or apprehensive but movements not aggressive or vigorous
0	Alert and calm	Spontaneously pays attention to caregiver
-1	Drowsy	Not fully alert, but has sustained (more than 10 seconds) awakening, with eye contact, to voice
-2	Light sedation	Briefly (less than 10 seconds) awakens with eye contact to voice
-3	Moderate sedation	Any movement (but no eye contact) to voice
-4	Deep sedation	No response to voice, but any movement to physical stimulation
-5	Unarousable	No response to voice or physical stimulation

https://doi.org/10.1371/journal.pone.0207174.t001

	DAY 0	DAY 7
RAAS score		

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PATIENT IS DELIRIOUS

CAM-ICU Worksheet

Feature 1: Acute Onset or Fluctuating Course	Score	Check here if Present
Is the pt different than his/her baseline mental status? OR Has the patient had any fluctuation in mental status in the past 24 hours as evidenced by fluctuation on a sedation scale (i.e., RASS), GCS, or previous delirium assessment?	Either question Yes →	☐
Feature 2: Inattention		
Letters Attention Test (See training manual for alternate pictures) Directions: Say to the patient, "I am going to read you a series of 10 letters. Whenever you hear the letter 'A,' indicate by squeezing my hand." Read letters from the following letter list in a normal tone 3 seconds apart. S A V E A H A A R T Errors are counted when patient fails to squeeze on the letter "A" and when the patient squeezes on any letter other than "A."	Number of Errors > 2 →	☐
Feature 3: Altered Level of Consciousness		
Present if the Actual RASS score is anything other than alert and calm (zero)	RASS anything other than zero →	☐
Feature 4: Disorganized Thinking		
Yes/No Questions (See training manual for alternate set of questions) 1. Will a stone float on water? 2. Are there fish in the sea? 3. Does one pound weigh more than two pounds? 4. Can you use a hammer to pound a nail? Errors are counted when the patient incorrectly answers a question. Command Say to patient: "Hold up this many fingers" (hold 2 fingers in front of patient). "Now do the same thing with the other hand" (do not repeat number of fingers). If patient is unable to move both arms, for second part of command ask patient to "Add one more finger." An error is counted if patient is unable to complete the entire command.	Combined number of errors > 1 →	☐

Overall CAM-ICU Feature 1 plus 2 and either 3 or 4 present = CAM-ICU positive		
	Criteria Met →	☐ CAM-ICU Positive (Delirium Present)
	Criteria Not Met →	☐ CAM-ICU Negative (No Delirium)

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	DAY 0	DAY 7
CAM ICU		

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PT IS CONSCIOUS

Mini-Mental State Examination (MMSE)

Patient's Name: _____ Date: _____

Instructions: ***Ask the questions in the order listed. Score one point for each correct response within each question or activity.***

Maximum Score	Patient's Score	Questions
5		"What is the year? Season? Date? Day of the week? Month?"
5		"Where are we now: State? County? Town/city? Hospital? Floor?"
3		The examiner names three unrelated objects clearly and slowly, then asks the patient to name all three of them. The patient's response is used for scoring. The examiner repeats them until patient learns all of them, if possible. Number of trials: _____
5		"I would like you to count backward from 100 by sevens." (93, 86, 79, 72, 65, ...) Stop after five answers. Alternative: "Spell WORLD backwards." (D-L-R-O-W)
3		"Earlier I told you the names of three things. Can you tell me what those were?"
2		Show the patient two simple objects, such as a wristwatch and a pencil, and ask the patient to name them.
1		"Repeat the phrase: 'No ifs, ands, or buts.'"
3		"Take the paper in your right hand, fold it in half, and put it on the floor." (The examiner gives the patient a piece of blank paper.)
1		"Please read this and do what it says." (Written instruction is "Close your eyes.")
1		"Make up and write a sentence about anything." (This sentence must contain a noun and a verb.)
1		"Please copy this picture." (The examiner gives the patient a blank piece of paper and asks him/her to draw the symbol below. All 10 angles must be present and two must intersect.)
30		TOTAL

(Adapted from Rovner & Folstein, 1987)

Source: www.medicine.uiowa.edu/igec/tools/cognitive/MMSE.pdf

1

Provided by NHCQF, 0106-410

	DAY 0	DAY 7
MMSE		

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ALTERATIONS IN CEREBRAL FUNCTION

		DAY 0	DAY 7
Concentration, fatigue	Serial seven test		
Myoclonus/twitching	Yes /No		
Delusion/hallucination	Yes /No		
Posture			
Asterixis	Yes /No		
Seizure	Yes /No If yes -Type		
Sleep inversion	Yes /No		
aphasia	Yes /No		

CRANIAL NERVES INVOLVEMENT

		Day 0	Day 7
CN1	Sense of smell		
CN2	Visual field-quadrant method Vision Pupils		
CN3,4,6	Nystagmus Extraocular movements		
CN5	Chew/bite/lateral jaw movements Sensory function		
CN 7	Motor'-smile/frown /raise eyebrows		
CN8	Rinne test Weber test		
CN 9,10	Gag reflex Swallowing Uvula movement		
CN 11	Turning head Upward shoulder shrug		
CN 12	Tongue movement		

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MUSCULAR DISORDERS

		Day 0	Day 7
Power	GRADING ICMR Upper limb R/L Lower limb R/L Trunk		
Tremor/involuntary movements	Yes/No		
Wasting	Yes/No		
Fasciculation	Yes/No		

SENSORY SYSTEM

	DAY 0	DAY 7
Touch, pin prick sensation,vibration		
vibration		
Stereognosis		
2 point discrimination		

DEEP TENDON REFLEX

Reflexes	Day 0	Day 7
Jaw jerk		
Triceps		
Supinator		
Knee		
Ankle		
Abdominal		
Plantar		

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Postural fainting-YES/NO		
Loss of urinary control -YES/NO		
Night diarrhea-YES/NO		

	Day 0	Day 7
Neck rigidity-yes/no		
Gait abnormality yes/no		

ADDITIONAL INVESTIGATIONS AS AND WHEN INDICATED

CT HEAD	
MRI	
EEG	
NCV/EMG	

CSF ANALYSIS- as and when required

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KEY TO MASTER CHART

R/E	:	ROUTINE EXAMINATION
UPCR	:	URINE PROTEIN CREATININE RATIO
Y	:	YES
N	:	NO
X	:	NOT DONE
-	:	LEFT AGAINST MEDICAL ADVICE

S.NO	Name	Age	Sex	Presenting complaints	ICU STAY	hypertension	diabetes mellitus 2	CLD	CAD	Any nephrotoxic drugs prior to admission	Type of nephrotoxic drugs	H/O Alcohol/smoking/substance abuse	Mechanical ventilatory support	Ionotropic support	qSOFA _Day 0	qSOFA _Day 7	CAUSE	AKI etiology	sepsis present	septic shock present	source of sepsis	UREA DAY 0
1	HS	61	M	CONSTIPATION	Y	N	N	N	Y	N		N	N	N	0	0	SURGICAL	INTESTINAL OBTSRUCTION	N	N		35
2	LC	90	F	PALPITATIONS	Y	Y	Y	N	Y	N		N	N	N	0	0	CARDIORENAL	CARDIORENAL	N	N		64
3	KS	76	M	DYSPNEA COUGH	Y	N	N	N	N	N		ALCOHOL	N	N	1	0	INFECTION	PNEUMONIA	N	N		52
4	SK	78	F	DYSPNEA COUGH	N	N	N	N	N	N		N	N	N	0		INFECTION	PNEUMONIA	N	N		71
5	DK	60	F	IRRITABILITY	N	Y	N	N	N	N		N	N	N	1	0	MISC	HYPERCALCEMIA	N	N		165
6	RM	17	M	VOMITING	N	N	N	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		35
7	SK	68	M	DYSPNEA COUGH	N	Y	N	N	N	N		SMOKING	N	N	1	0	INFECTION	PNEUMONIA	N	N		31
8	R	24	F	VOMITING	Y	N	N	N	N	N		N	N	N	0	0	INFECTION	VIRAL HEPATITIS	N	N		43
9	DR	75	M	FEVER	N	Y	N	N	N	N		N	N	N	0	0	TROPICAL FEVER	DENGUE	N	N		100
10	SK	49	M	VOMITING	N	N	N	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		50
11	TS	29	M	FEVER	N	N	N	N	N	N		N	N	N	0	0	TROPICAL FEVER	DENGUE	N	N		40
12	NK	38	M	DYSPNEA COUGH	N	N	N	N	N	N		N	N	N	0	0	INFECTION	PNEUMONIA	N	N		117
13	MK	31	M	LOOSE STOOLS	N	N	N	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		36
14	R	46	F	PAIN ABDOMEN	N	N	N	N	N	N		N	N	N	0	0	INFECTION	UTI	N	N		63
15	BR	70	M	PAIN ABDOMEN	N	Y	Y	Y	Y	N		N	N	N	0	0	INFECTION	PYELONEPHRITIS	N	N		94
16	D	23	M	LOOSE STOOLS	N	N	N	N	N	Y	ALTERNATIVE MEDICATION	N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		62
17	MA	19	M	VOMITING	N	N	N	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		45
18	BS	35	M	ALTERED SENSORIUM	N	N	Y	N	N	N		N	N	Y	2		INFECTION	UTI	Y	Y	UROSEPSIS	57
19	HK	52	M	FEVER	N	N	Y	N	N	N		N	N	N	0	0	TROPICAL FEVER	DENGUE	N	N		83
20	RL	50	M	LOOSE STOOLS	N	N	Y	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		65
21	R S	21	M	LOOSE STOOLS	N	N	N	N	N	N		ALCOHOL	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		46
22	JK	66	F	DYSPNEA COUGH	Y	N	Y	N	N	N		N	N	N	1	0	INFECTION	PNEUMONIA	N	N		80
23	BK	75	F	BURNING MICTURITION	N	Y	Y	N	Y	N		N	N	N	0	0	INFECTION	UTI	N	N		69
24	SK	53	F	VOMITING	N	Y	Y	N	N	N		N	N	Y	1	0	INFECTION	ACUTE GASTROENTERITIS	N	N		67
25	RK	63	F	UNCONTROLLED HYPERTENSION	N	Y	N	N	N	N		N	N	N	0	0	CARDIORENAL	HYPERTENSIVE URGENCY	N	N		53
26	VK	48	M	ABDOMINAL DISTENSION	N	N	N	Y	N	N		N	N	N	0	0	HRS	HRS	N	N		112
27	US	61	F	FEVER	Y	Y	Y	N	N	N		N	N	N	0		INFECTION	PACHYMENINGITIS, PNEUMONIA	N	N		57
28	US	57	M	LOOSE STOOLS	N	Y	N	N	N	N		N	N	N	0		INFECTION	ACUTE GASTROENTERITIS	N	N		36
29	VKS	71	M	BURNING MICTURITION	Y	N	Y	N	Y	N		N	N	N	0	0	INFECTION	UTI	N	N		57
30	BKS	75	M	PAIN ABDOMEN	N	Y	Y	N	Y	N		N	N	N	0	0	INFECTION	PYELONEPHRITIS	N	N		80
31	KR	67	F	VOMITING	N	Y	Y	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		41
32	RK	40	M	PAIN ABDOMEN	N	N	Y	N	N	N		ALCOHOL	N	N	0	0	PANCREATITIS	ACUTE PANCREATITIS	N	N		46
33	JK	75	F	LOOSE STOOLS	N	Y	N	N	Y	N		N	N	Y	1	0	INFECTION	ACUTE GASTROENTERITIS	N	N		161
34	KK	62	F	FEVER	N	N	N	N	N	N		N	N	N	0		TROPICAL FEVER	DENGUE	N	N		149
35	KS	74	F	DECREASED APPETITE	N	Y	Y	N	N	N		N	N	N	0	0	INFECTION	UTI	N	N		94
36	GS	52	M	PAIN IN EYE	N	N	Y	N	N	N		N	N	N	0		INFECTION	ORBITAL CELLULITIS	N	N		86
37	GS	68	M	DYSPNEA COUGH	N	Y	Y	N	Y	N		N	N	N	0	0	CARDIORENAL	CARDIORENAL	N	N		63
38	SK	58	M	PAIN ABDOMEN	N	N	Y	N	N	N		ALCOHOL	N	N	0		PANCREATITIS	ACUTE PANCREATITIS	N	N		134
39	BK	81	F	VOMITING	N	N	Y	N	Y	N		N	N	N	0	0	MISC	DIABETIC KETOACIDOSIS	N	N		114
40	LK	56	F	PAIN ABDOMEN	N	N	Y	N	N	N		N	N	N	0	0	OBSTRUCTIVE	NEPHROLITHIASIS	N	N		43
41	BS	53	M	FEVER ALTERED SENSORIUM	Y	N	N	N	N	N		IV DRUGS	Y	Y	3	3	INFECTION	INFECTIVE ENDOCARDITIS	Y	Y	INFECTIVE ENDOCARDITIS	96
42	N	56	F	PAIN ABDOMEN	N	N	Y	N	N	N		N	N	N	0	0	INFECTION	ACUTE CHOLANGITIS	N	N		46
43	KD	70	F	LOOSE STOOLS	N	Y	N	N	N	N		N	N	Y	1	0	INFECTION	ACUTE GASTROENTERITIS	N	N		183
44	VPP	76	M	CHEST PAIN	N	Y	Y	N	Y	N		SMOKING	N	N	0		CARDIORENAL	CARDIORENAL	N	N		54
45	AR	81	M	FEVER	N	Y	Y	N	N	N		N	N	N	0		TROPICAL FEVER	DENGUE	N	N		161
46	RS	69	M	ABNORMAL BODY MOVEMENTS	Y	Y	N	N	Y	N		N	N	N	1	1	INFECTION	MENINGITIS	N	N		58
47	SS	26	M	YELLOWING OF SCLERA	N	N	N	Y	N	N		N	N	N	0	0	INFECTION	VIRAL HEPATITIS	N	N		112

S.NO	Name	UREa day 7	Serum creatinine	CREAT DAY 7	AKI STAGE	Dialysis session	SBP_DAY 0	DBP_DAY 0	SBP_DAY 7	DBP_DAY 7	Heart rate DAY 0	Heart rate DAY 7	Respiratory rate DAY 0	Respiratory rate DAY 7	Saturation oxygen DAY 0	Saturation oxygen DAY 7	TLC DAY 0	TLC DAY 7	Platelet count DAY 0	Platelet count DAY 7	Sodium DAY 0	Sodium DAY 7	Potassium DAY 0	Potassium DAY 7
1	HS	143	2.1	8	1	Y	100	78	122	86	99	84	18	16	92	94	31	28	287	222	140	147	3.8	3.4
2	LC	65	1.9	1.3	1	N	112	82	116	82	132	122	18	14	90	94	5	4	49	35	132	130	3.9	4.9
3	KS	68	2	1.4	1	N	133	88	128	74	88	92	22	18	88	90	24	11	128	147	137	134	4.1	3.3
4	SK	32	1.7	0.9	1	N	124	78	132	78	76	84	18	16	96	97	21	12	122	154	146	140	3.8	4.2
5	DK	109	5.4	1.6	3	N	118	88	130	88	70	68	16	18	96	94	11	6	358	248	122	137	4.4	3.07
6	RM	28	2.8	0.8	2	N	116	74	118	84	88	84	16	14	95	94	12	8	375	322	135	137	4.7	4.1
7	SK	29	1.8	1	1	N	132	78	128	84	99	78	22	14	90	94	12	7	293	255	133	139	4.18	3.2
8	R	25	2.3	1	2	N	100	70	122	84	68	74	18	16	93	95	8	6	188	178	137	139	4.1	4.1
9	DR	74	3.6	2.4	3	N	122	74	114	74	89	78	14	14	98	94	10	8	132	122	134	141	5.9	3.6
10	SK	24	1.6	1.2	1	N	108	68	122	85	98	82	14	14	95	97	11	9	202	198	144	142	2.9	3.8
11	TS	26	1.4	1.1	1	N	108	68	122	84	72	68	16	14	94	97	1.9	3.7	26	46	145	141	4.2	4.5
12	NK	84	3.5	1.8	3	N	122	78	118	82	82	78	18	14	92	95	11	8	318	252	129	139	4.9	4.1
13	MK	24	2.9	1.1	2	N	128	75	132	88	98	75	14	16	95	93	19	12	457	358	133	138	5.1	4.8
14	R	9	2.3	0.5	2	N	128	78	122	84	84	82	14	16	95	93	12	8	208	204	141	140	3.6	3.1
15	BR	125	2.4	2.1	2	N	110	78	122	86	74	85	14	16	94	96	10	8	62	68	142	139	4.2	3.3
16	D	34	4.9	0.9	3	N	122	80	128	84	80	82	16	18	95	94	13	8	284	222	136	139	4.7	3.9
17	MA	32	2.1	0.9	1	N	133	80	128	78	94	90	16	14	96	97	11	8	100	112	137	135	4.6	4.3
18	BS		1.9		1	N	72	54			95		14		92		18		145		134		5.2	
19	HK	46	2.1	1.8	1	N	112	84	122	86	88	74	16	14	95	97	5	7	32	64	129	134	4.5	4.1
20	RL	34	1.7	0.9	1	N	122	78	132	84	96	78	14	16	95	96	7	5	96	74	142	135	6.6	4.1
21	R S	31	2.9	1	2	N	128	74	118	76	98	92	18	16	95	94	14	11	142	132	135	138	5.2	3.7
22	JK	55	1.6	1.06	1	N	133	74	128	72	74	86	24	18	88	94	10	8	172	164	133	136	4.5	4
23	BK	69	1.9	1.5	1	N	116	74	128	74	89	76	16	14	93	95	12	11	290	255	127	135	4.6	4.8
24	SK	15	2.1	0.6	1	N	78	56	110	74	100	84	14	16	92	96	11	8	140	122	128	145	4.6	3.3
25	RK	82	1.8	2	1	N	180	100	132	78	78	76	16	14	95	94	10	6	130	128	136	131	4.3	3.01
26	VK	73	1.6	1	1	N	102	68	110	74	88	86	16	14	95	94	4	3	62	58	135	134	4.6	4.1
27	US		1.7		1	N	100	72			89				95		15		160		130		2.9	
28	US	22	2.9	0.8	2	N	100	74	122	82	122	114	18	14	96	94	17	8	326	228	135	132	6.6	4.1
29	VKS	54	1.7	1.4	1	N	116	78	122	76	102	100	18	16	96	94	6	4	181	178	136	139	4.8	4.6
30	BKS	43	2.5	0.9	2	N	112	70	122	74	112	84	16	14	93	95	9	7	189	158	128	135	4.6	4.4
31	KR	36	1.6	1.2	1	N	128	78	132	92	98	94	16	18	95	97	8.5	7	292	255	135	140	4.5	4.4
32	RK	29	1.6	0.6	1	N	112	78	128	84	94	88	16	14	90	94	12	10	212	188	129	130	6.9	3.7
33	JK	100	4.8	2.1	3	Y	74	58	100	78	84	75	18	12	96	94	18	14	128	118	131	135	4.2	3.8
34	KK	110	4.9	1.9	3	N	110	74	133	82	72	92	16	14	90	92	18	14	22	55	139	136	6.9	4.3
35	KS	106	2.1	0.9	1	N	122	78	118	86	89	78	14	16	88	90	15	11	189	178	126	134	4.8	4.1
36	GS	32	1.3	0.8	1	N	148	80	132	82	98	88	16	14	94	96	9	7	213	200	140	141	4.5	4.1
37	GS	45	1.7	1.5	1	N	118	78	106	84	112	78	16	18	93	94	11	8	158	144	128	135	3.2	3.8
38	SK		3.5		3	N	100	78			102		18		93		13		90		147		3.9	
39	BK	72	3.3	1.8	3	N	98	76	94	75	89	75	14	16	94	97	15	8	126	145	144	150	3.3	4.5
40	LK	34	1.6	0.8	1	N	132	84	144	92	90	89	16	14	96	95	7	8	245	267	146	135	3.8	3.4
41	BS	70	2.1	1.1	1	N	76	54	88	68	112	102			92	93	32	8	83	90	136	138	4.5	4.1
42	N	31	1.6	0.9	1	N	132	78	144	84	94	86	14	16	95	96	8	6	78	84	141	138	4.14	3.8
43	KD	196	4.3	5.01	3	Y	110	74	112	74	88	98	16	14	91	93	19	11	66	54	141	135	4.6	4.1
44	VPP	23	1.6	0.7	1	N	100	70	122	84	78	84	16	14	93	95	7	4	202	188	140	133	3.9	4.1
45	AR	54	3	1.1	2	N	114	76	118	82	88	76	14	16	92	96	3.4	5	100	174	130	142	7	5
46	RS	32	1.7	0.9	1	N	118	74	122	86	84	76	16	14	94	95	7	8	124	115	147	135	4	5.1
47	SS	88	1.7	0.9	1	N	122	74	118	76	99	92	16	14	95	94	11	8	221	128	138	131	3.7	3.3

S.NO	Name	AST DAY 0	AST DAY 7	ALT DAY 0	ALT DAY 7	ALP DAY 0	ALP DAY 7	Bilirubin DAY 0	Bilirubin DAY 7	Total protein DAY 0	Total protein DAY 7	Albumin DAY 0	Albumin DAY 7	Calcium DAY 0	Parathyroid (if done)	Urine R/E	UPCr(IF DONE)	Urine culture	Blood culture	Any other culture	NEUROLOGICAL FEATURES yes/no	GCS DAY 0	GCS DAY 7	Scenario 1- pt is sedated	RAAS score DAY 0
1	HS	85	80	91	90	139	137	0.3	0.1	11	10	6.2	5.8	11.5		PROTEINURIA		N	N		N	15	15	N	
2	LC	44	40	25	20	103	112	0.3	0.4	7.4	7.3	3.8	3.7	7.8		NORMAL		N			N	15	15	N	
3	KS	45	42	22	20	64	60	0.8	0.7	5.9	5.7	3.4	3.2	9.8		RBC	0.78				N	15	15	N	
4	SK	64	32	54	28	80	55	0.9	0.4	6.9	6.1	2.8	2.7	9		NORMAL		N			N	15	15	N	
5	DK	43	41	50	62	112	98	1.1	0.9	6.8	6.2	2.7	2.6	13	76	NORMAL	0.9	N	N		Y	13	15	N	
6	RM	48	42	50	54	233	114	0.4	0.2	10	8	6.8	5	8.3		NORMAL					N	15	15	N	
7	SK	22	20	13	11	63	60	0.17	0.1	6	5.8	3.45	3.3	9.1		NORMAL					N	14	15	N	
8	R	4412	1183	5839	3045	224	194	3	4	5.9	5.2	3.4	2.8	8		NORMAL					N	15	15	N	
9	DR	548	122	455	112	179	164	0.3	0.2	6.1	5.8	3.3	3.1	7.9		PROTEINURIA		N	N		N	15	15	N	
10	SK	25	22	25	24	79	86	0.2	0.1	5.4	4.8	2.7	2.6	9		NORMAL					N	15	15	N	
11	TS	75	68	32	28	75	58	0.3	0.2	6.4	6.2	4	2.9	9.1		NORMAL			N		N	15	15	N	
12	NK	74	45	78	42	112	135	1.1	0.9	7.4	6.8	2.8	2.4	9.1		NORMAL		N			N	15	15	N	
13	MK	47	40	43	38	161	154	0.7	0.5	11	8.8	6.7	5.4	10.8		NORMAL		N	N		N	15	15	N	
14	R	19	17	20	18	115	112	0.1	0.1	8.6	7.4	3.7	3.9	8.4		NORMAL		N			N	15	15	N	
15	BR	60	54	38	32	661	588	2.1	1.8	5.2	5.8	2.3	2.1	8.4		PUS CELLS		N	N		N	15	15	N	
16	D	65	45	18	14	153	110	0.3	0.2	8	7	5.2	4.8	8.6		NORMAL					N	15	15	N	
17	MA	46	58	28	45	153	112	0.6	0.4	9	7	5.8	3.7	9.2		NORMAL					N	15	15	N	
18	BS	22		18		118		0.3		6.5		3.9		9.1		PROTEINURIA		ENTEROCOCCUS	N	SPUTUM-KLEBSIELLA	Y	10		N	
19	HK	55	32	68	64	336	112	1.7	0.8	6.1	5.8	3.3	3.1	8.4		NORMAL					N	15	15	N	
20	RL	73	34	122	85	66	54	1.2	0.8	5.7	5.1	3.4	3.1	8		NORMAL					N	15	15	N	
21	R S	124	112	343	312	165	102	0.9	0.7	10	8	6.7	4.8	10.8		NORMAL		N	N		N	15	15	N	
22	JK	41	38	66	58	73	62	0.8	0.5	5.4	5.2	2.8	2.4	8.9		PUS CELLS		N	N		N	15	15	N	
23	BK	30	22	8	10	111	108	0.6	0.4	7.4	7	3.4	3.1	8.4		PUS CELLS		ENTEROCOCCUS , SERRATIA			N	15	15	N	
24	SK	42	40	57	50	90	88	0.6	0.5	5.4	6	2.9	2.8	10		RBC					N	15	15	N	
25	RK	24	18	17	16	123	118	0.3	0.1	8	7	4.8	4.2	8.7		NORMAL		N	N		N	15	15	N	
26	VK	86	78	62	58	100	98	2.1	0.9	7	6	3.3	2.8	8.1		NORMAL		N	N		N	15	15	N	
27	US	78		40		90		0.9		8.1		4.4		9.2		NORMAL		N	N		Y	6		Y	-2
28	US	41	32	36	28	113	122	0.7	0.4	10	7.1	6.4	4.2	10		PROTEINURIA					N	15	15	N	
29	VKS	38	28	32	28	58	54	0.7	0.5	6.4	5.8	3.6	3.2	9		NORMAL					N	15	15	N	
30	BKS	223	59	200	120	259	277	5.6	3.3	5.4	5.3	2.7	2.8	9		PUS CELLS		N	N		N	15	15	N	
31	KR	26	24	16	14	105	100	0.3	0.1	8	7	2.16	2	8.9		PROTEINURIA	7				N	15	15	N	
32	RK	51	48	33	28	126	155	1.9	0.9	6.8	6.7	3.5	2.8	7.4		NORMAL		N	N		N	15	15	N	
33	JK	48	42	35	32	58	54	0.4	0.1	6	5.8	2.9	2.8	9		NORMAL					N	15	15	N	
34	KK	469	212	148	100	234	112	3.8	1.2	5.5	6	2.4	2.8	8.1		NORMAL					N	15	15	N	
35	KS	24	22	13	12	140	100	0.4	0.4	6.9	6.2	2.8	2.4	8		PROTEINURIA		E COLI	N		N	15	15	N	
36	GS	58	42	54	38	112	66	0.3	0.2	6.7	6.3	2.6	2.8	7		NORMAL					N	15	15	N	
37	GS	88	54	92	45	112	102	1	0.8	6.4	5.8	3.2	3.4	9.2		PROTEINURIA		N	N		N	15	15	N	
38	SK	117		49		91		9.3		5.5		2.8		7.8		PUS CELLS		N	N		N	15	15	N	
39	BK	45	38	56	40	80	72	0.9	0.4	6.8	5.9	2.7	2.6	9		PROTEINURIA					N	15	15	N	
40	LK	19	14	14	18	104	132	1.6	0.9	7	6.4	4.5	3.4	8.8		RBC		N			N	15	15	N	
41	BS	6768	348	4546	348	117	89	3.4	2.3	6.4	4.9	3.9	3	8.8		PUS CELLS		N	N		Y	8	6	Y	-2
42	N	32	25	36	22	1094	2248	4.6	2.8	5.3	5.6	2.01	2.6	8.6		NORMAL		N	N		N	15	15	N	
43	KD	21	12	9	14	65	48	0.3	0.2	5.8	5.5	3.07	2.8	9.1		RBC	1.1	N	N		N	15	15	N	
44	VPP	17	13	69	34	69	28	0.6	0.2	6.8	6.5	3.7	3.1	8		NORMAL	0.5				N	15	15	N	
45	AR	286	122	101	52	92	42	2.2	1.8	6.3	7.1	2.3	2.9	8.2		NORMAL					N	15	15	N	
46	RS	45	32	84	32	112	138	1.1	0.9	6.4	6.1	2.3	2.8	9.7		NORMAL		N	N		Y	10	13	N	
47	SS	5731	1202	4981	1600	141	100	20	35	7	5.9	4.2	3.7	9		RBC					N	15	15	N	

S.NO	Name	RAAS score DAY 7	PATIENT IS DELIRIOUS	CAM ICU DAY 0	CAM ICU DAY 7	MMSE DAY 0	MMSE DAY 7	SERIAL 7_DAY 0	SERIAL 7 _DAY 7	MYCOLONUS DAY 0	MYCOLONUS DAY 7	DELUSION/HALLUCINATION DAY 0	DELUSION/HALLUCINATION DAY 7	Asterixis DAY 0	Asterixis DAY 7	Seizure DAY 0	Seizure DAY 7	Sleep inversion DAY 0	Sleep inversion DAY 7	aphasia DAY 0	aphasia DAY 7	cranial nerve involved	MUSCULAR DISORDERSDAY 0	MUSCULAR DISORDERS DAY 7	Tremor/involuntary movements DAY 0
1	HS		N	N	N	27	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
2	LC		N	N	N	26	27	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
3	KS		N	N	N	25	26	5	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
4	SK		N	N	N	25	26	5	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
5	DK		Y	Y	N	0	22	0	2	N	N	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N
6	RM		N	N	N	26	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
7	SK		N	N	N	26	25	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
8	R		N	N	N	26	26	2	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
9	DR		N	N	N	27	28	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
10	SK		N	N	N	26	26	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
11	TS		N	N	N	27	27	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
12	NK		N	N	N	28	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
13	MK		N	N	N	27	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
14	R		N	N	N	28	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
15	BR		N	N	N	26	26	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
16	D		N	N	N	27	26	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
17	MA		N	N	N	26	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
18	BS		Y	Y		0		0		Y		N		N		N		N		N		N	N		Y
19	HK		N	N	N	26	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
20	RL		N	N	N	27	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
21	R S		N	N	N	28	29	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
22	JK		N	N	N	25	26	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
23	BK		N	N	N	26	26	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
24	SK		N	N	N	26	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
25	RK		N	N	N	26	25	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
26	VK		N	N	N	27	26	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
27	US					0		0		N		N		N		Y		N		N		N	N		N
28	US		N	N	N	27	28	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
29	VKS		N	N	N	26	28	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
30	BKS		N	N	N	27	28	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
31	KR		N	N	N	26	27	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
32	RK		N	N	N	26	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
33	JK		N	N	N	28	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
34	KK		N	N	N	26	27	5	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
35	KS		N	N	N	27	28	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
36	GS		N	N	N	27	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
37	GS		N	N	N	28	27	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
38	SK		N	N	N	27	26	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
39	BK		N	N	N	27	26	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
40	LK		N	N	N	24	25	5	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
41	BS	-4	N	N	N	0	0	0	0	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
42	N		N	N	N	26	26	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
43	KD		N	N	N	25	26	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
44	VPP		N	N	N	26	27	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
45	AR		N	N	N	27	26	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
46	RS		Y	Y	N	0	0	0	0	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
47	SS		N	N	N	27	28	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N

S.NO	Name	Tremor/involuntary movements DAY 7	Wasting DAY 0	Wasting DAY 7	Fasciculation DAY 0	Fasciculation DAY 7	SENSORY SYSTEM	ABNORMAL DEEP TENDON reflexes	LOSS OF URINARY CONTROL DAY 0	LOSS OF URINARY CONTROL DAY 7	Night diarrhea DAY 0	Night diarrhea DAY 7	Neck rigidity DAY 0	Neck rigidity DAY 7	ADDITIONAL INVESTIGATIONS AS AND WHEN INDICATED_CT HEAD	MRI	EEG	CSF ANALYSIS-as and when required	USG abdomen
1	HS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PROMINENT GUT LOOPS
2	LC	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CHOLELITHIASIS
3	KS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
4	SK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
5	DK	N	N	N	N	N	X	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
6	RM	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
7	SK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
8	R	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HEPATITIS
9	DR	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB EDEMA
10	SK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
11	TS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB EDEMA
12	NK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
13	MK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CHOLELITHIASIS
14	R	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CYSTITIS
15	BR	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CYSTITIS,PERINEPHRIC STRANDING
16	D	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
17	MA	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
18	BS		N		N		X	N	N		N		N		NORMAL	X	X	X	PYELITIS
19	HK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB EDEMA
20	RL	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
21	R S	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PUS CELLS
22	JK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PERINEPHRIC STRANDING
23	BK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CYSTITIS
24	SK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ACUTE RENAL DISEASE
25	RK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CHOLELITHIASIS
26	VK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ASCITES, CIRRHOSIS
27	US		N		N		X	N	N		N		Y		NORMAL	PACHYMENINGITIS	X	TLC-2 NORMAL GLUCOSE PROTEIN AND CHLORIDE	CHOLELITHIASIS
28	US	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
29	VKS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
30	BKS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PERINEPHRIC STRANDING
31	KR	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB EDEMA
32	RK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PANCREATITIS, BULKY HEAD
33	JK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER, CHOLELITHIASIS
34	KK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB EDEMA
35	KS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CYSTITIS
36	GS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
37	GS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
38	SK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PANCREATITIS
39	BK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
40	LK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	RENAL CALCULI
41	BS	N	Y	Y	N	N	X	N	N	N	N	N	Y	N	X	ANGIOINVASIVEFUNGALINFECTIOM/ MYCOTIC	X	HIGHPROTEIN-117MNC-PREDOMINANT	FATTY LIVER
42	N	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CHOLELITHIASIS, CHOLANGITIS
43	KD	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
44	VPP	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
45	AR	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB EDEMA FATTY LIVER
46	RS	N	N	N	N	N	X	N	N	N	N	N	Y	N	NORMAL	X	X	HIGH PROTEIN NORMAL GLUCOSE AND TLC-2	SPLENOMEGALY
47	SS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HEPATITIS ASCITES

S.NO	Name	Age	Sex	Presenting complaints	ICU STAY	hypertension	diabetes mellitus 2	CLD	CAD	Any nephrotoxic drugs prior to admission	Type of nephrotoxic drugs	H/O Alcohol/smoking/substance abuse	Mechanical ventilatory support	Ionotropic support	qSOFA _Day 0	qSOFA _Day 7	CAUSE	AKI etiology	sepsis present	septic shock present	source of sepsis	UREA DAY 0
48	BK	65	F	DECREASED AROUSAL	Y	Y	Y	N	Y	N		N	N	Y	1		INFECTION	UTI	N	N		202
49	HS	61	M	LOOSE STOOLS	N	N	N	N	Y	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		66
50	AS	74	M	CONSTIPATION	Y	Y	N	N	N	Y	OPIOD	OPIOD	N	N	1	0	SURGICAL	INTESTINAL OBTSRUCTION	N	N		144
51	BS	85	M	DECREASED URINE OUTPUT	N	Y	Y	N	N	N		N	N	N	2	0	INFECTION	DIABETIC FOOT	Y	N	DIABETIC FOOT	90
52	SK	45	F	YELLOWING OF EYES	Y	N	Y	N	N	N		N	N	N	0	0	MISC	HEMOLYTIC UREMIC SYNDROME	N	N		98
53	NS	52	M	DECREASED AROUSAL	Y	Y	N	N	N	N		N	N	N	2	1	INFECTION	PNEUMONIA	N	N		202
54	KJ	78	M	BURNING MICTURITION	N	N	Y	N	N	N		N	N	N	0	0	INFECTION	UTI	N	N		105
55	GS	75	M	BURNING MICTURITION	N	N	Y	N	N	N		N	N	N	0	0	INFECTION	UTI	N	N		143
56	GS	30	M	SNAKE BITE	Y	N	N	Y	N	N		ALCOHOL	N	N	1		MISC	SNAKE BITE	N	N		113
57	RK	58	F	LOOSE STOOLS	N	Y	N	N	N	N		N	N	N	0	0	INFECTION	ACUTE CHOLECYSTITIS	N	N		66
58	B	64	F	FEVER	Y	Y	N	N	N	N		N	N	Y	2	1	MISC	HYPERCALCEMIA	N	N		149
59	VKK	76	M	FEVER COUGH	N	Y	N	N	Y	N		N	N	N	1	0	INFECTION	PNEUMONIA	N	N		143
60	BS	58	M	FEVER	N	N	Y	N	N	N		N	N	N	0	0	TROPICAL FEVER	DENGUE	N	N		167
61	PKJ	70	M	LOOSE STOOLS	Y	N	N	N	N	N		N	N	N	1	0	INFECTION	ACUTE GASTROENTERITIS	N	N		55
62	PS	78	M	VOMITING	Y	Y	Y	N	N	N		#NAME?	N	Y	2	0	INFECTION	ACUTE CHOLANGITIS	Y	Y	HEPATOPANCREATOBILIARY	122
63	N	63	F	PAIN ABDOMEN	N	N	N	N	N	Y	VITAMIN D	N	N	N	1	0	MISC	HYPERCALCEMIA	N	N		40
64	OP	70	M	DYSPNEA COUGH	N	N	Y	N	N	N		N	N	N	0	0	INFECTION	PNEUMONIA	N	N		184
65	UK	75	F	LOOSE STOOLS	N	Y	N	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		127
66	HS	55	M	CHEST PAIN	N	Y	Y	N	Y	N		N	N	N	0	0	CARDIORENAL	CARDIORENAL	N	N		85
67	SS	62	F	BURNING MICTURITION	N	Y	Y	N	Y	N		N	N	N	0	0	INFECTION	UTI	N	N		77
68	N	74	M	PAIN ABDOMEN	N	Y	Y	N	Y	N		N	N	N	0	0	INFECTION	UTI	N	N		143
69	RA	57	M	ABDOMINAL DISTENSION	N	N	N	Y	N	N		ALCOHOL	N	N	1	0	INFECTION	SBP	N	N		102
70	RS	45	M	DYSPNEA COUGH	N	N	Y	N	N	N		ALCOHOL	N	N	0	0	INFECTION	PNEUMONIA	N	N		61
71	TR	65	M	LOOSE STOOLS	N	Y	N	N	N	N		N	N	Y	2	1	INFECTION	ACUTE GASTROENTERITIS	Y	N	GASTROENTEROLOGY	388
72	NR	65	F	FEVER ALTERED SENSORIUM	Y	Y	N	N	N	N		N	N	N	2		TROPICAL FEVER	MALARIA FALCIPARUM	N	N		143
73	N	50	F	ALTERED SENSORIUM	Y	N	N	N	N	N		N	N	N	1		MISC	HYPERCALCEMIA;MENINGITIS	N	N		179
74	SK	44	F	ALTERED SENSORIUM	Y	Y	Y	N	N	N		N	N	Y	2		INFECTION	UTI	Y	Y	UROSEPSIS	186
75	KS	66	M	FEVER ALTERED SENSORIUM	Y	Y	Y	N	N	N		ALCOHOL	N	N	2	1	INFECTION	TUBERCULOSIS	N	N		70
76	B	48	M	VOMITING	N	N	N	N	N	N		N	N	N	0	0	TROPICAL FEVER	LEPTOSPIRA	N	N		138
77	ASN	49	M	LOOSE STOOLS	N	N	N	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		68
78	BS	70	M	HEMOPTYSIS	N	Y	N	N	N	N		N	N	N	0	0	INFECTION	FUNGAL PNEUMONIA	N	N		48
79	SG	78	M	LOSS OF CONSCIOUSNESS, FEVER	Y	Y	N	N	N	N		N	Y	N	2	2	TROPICAL FEVER	COMPLICATED TROPICAL FEVER	N	N		72
80	AS	34	M	PAIN ABDOMEN	N	N	N	N	N	N		N	N	N	0	0	TROPICAL FEVER	LEPTOSPIRA	N	N		79
81	SR	58	F	MALAISE	N	Y	Y	N	N	N		N	N	N	0	0	MISC	DISSEMINATED TB, POLYMYOSITIS	N	N		66
82	M	29	F	ABNORMAL BODY MOVEMENTS	Y	N	N	N	N	N		N	Y	N	2	1	TROPICAL FEVER	DENGUE	N	N		128
83	BK	60	F	PAIN ABDOMEN	N	Y	N	N	Y	N		N	N	N	0	0	INFECTION	ACUTE CHOLECYSTITIS	N	N		174

S.NO	Name	UREa day 7	Serum creatinine	CREAT DAY 7	AKI STAGE	Dialysis session	SBP_DAY 0	DBP_DAY 0	SBP_DAY 7	DBP_DAY 7	Heart rate DAY 0	Heart rate DAY 7	Respiratory rate DAY 0	Respiratory rate DAY 7	Saturation oxygen DAY 0	Saturation oxygen DAY 7	TLC DAY 0	TLC DAY 7	Platelet count DAY 0	Platelet count DAY 7	Sodium DAY 0	Sodium DAY 7	Potassium DAY 0	Potassium DAY 7
48	BK		2.3		2	Y	110	74			95		22		90		16		199		132		3.8	
49	HS	112	3.3	6.5	3	N	144	78	132	76	88	76	16	14	97	96	11	8	132	122	140	148	3.8	4
50	AS	98	3.8	2.5	3	N	132	78	144	86	102	99	12	14	92	94	10	8	246	240	136	146	5.3	4.08
51	BS	48	2.1	1.4	1	N	84	50	124	84	99	74	22	16	94	95	15	11	212	252	138	144	5.8	4.8
52	SK	237	4.7	8	3	Y	110	78	122	86	99	74	16	14	93	94	34	24	24	18	141	140	4.2	4.3
53	NS	110	6	1.8	3	Y	110	78	126	84	99	76	26	18	86	90	3	15	409	388	135	147	6.3	4.8
54	KJ	45	5	0.9	3	N	133	86	128	74	84	80	14	16	95	97	17	14	205	194	133	136	3.17	4.1
55	GS	100	1.6	0.9	1	N	122	84	132	76	90	88	18	16	95	96	21	8	484	422	127	132	6	4.1
56	GS		3.5		3	Y	122	84			92		16		97		46		97		134		3.3	
57	RK	54	1.6	0.7	1	N	130	78	128	84	102	94	18	16	95	94	8	5	187	168	146	140	3.7	3.8
58	B	110	5.3	3.5	3	N	128	84	110	76	111	98	16	14	95	93	0.5	1.4	46	32	128	126	4	3.8
59	VKK	140	3	2.6	2	N	122	84	126	74	94	84	24	16	86	94	10	12	144	163	143	141	3.1	2.6
60	BS	84	3.6	1.2	3	N	126	78	112	74	94	98	16	14	96	97	14	8	200	155	133	128	4.7	4.1
61	PKJ	28	1.5	0.9	1	N	110	74	128	85	92	74	16	16	96	98	18	11	218	224	139	135	4.4	4
62	PS	154	3.4	3.9	3	N	82	54	112	86	122	98	16	14	96	94	21	16	235	185	118	119	5.2	3.6
63	N	36	1.5	0.9	1	N	133	84	122	80	68	74	14	16	96	94	14	8	198	176	133	143	3.9	3.7
64	OP	82	2.4	0.9	2	N	144	80	132	72	84	76	16	18	94	95	28	14	350	225	141	128	4.8	4.1
65	UK	74	2.9	1.4	2	N	132	87	122	74	74	70	14	16	95	90	9	6	79	74	138	131	4.08	3.35
66	HS	34	1.6	0.9	1	N	140	87	132	76	88	84	14	16	95	96	6	5.5	202	234	132	128	4.9	4.1
67	SS	60	1.7	1.2	1	N	118	78	124	86	72	88	14	18	95	97	14	8	130	188	138	141	4.1	3.8
68	N	114	2.1	1.7	1	N	133	84	141	92	80	84	14	16	94	98	64	22	100	145	135	134	4.9	4.18
69	RA	96	1.9	1.1	1	N	100	74	110	86	92	86	16	14	90	91	4.7	6.4	80	70	128	126	4.17	4
70	RS	35	1.8	0.9	1	N	136	75	128	80	84	75	14	16	94	97	14	8	158	188	128	137	4.9	3.4
71	TR	58	10	1.1	3	Y	84	56	132	84	88	76	22	16	94	96	14	8	502	244	133	150	6.6	4.2
72	NR		1.7		1	Y	140	98			89		22		88		7		31		128		6.3	
73	N		4.7		3	Y	102	74			84		16		95		18		99		138		4.5	
74	SK		4.8		3	Y	86	54			98		18		86		16		216		139		5.04	
75	KS	24	2	1.2	1	N	100	60	110	78	88	78	16	14	95	94	28	15	470	385	120	128	4.5	4.1
76	B	81	3.5	1.8	3	N	115	74	122	84	84	88	18	16	94	97	10	8	362	284	124	131	4.2	3.8
77	ASN	16	2	0.8	1	N	116	74	122	84	74	82	14	16	97	96	10	8	228	245	128	131	3.8	4.1
78	BS	29	1.8	1.3	1	N	122	78	132	74	88	89	14	16	96	94	14	11	148	138	141	148	4.1	4.2
79	SG	56	1.9	0.9	1	N	112	78	132	84	92	88	16	14	94	97	26	9	28	18	118	140	3.6	4.1
80	AS	45	2.9	2.3	2	N	132	78	122	84	94	92	18	16	95	96	6	7	140	110	138	137	3.9	4.1
81	SR	43	1.6	1.2	1	N	122	78	112	74	88	76	14	16	94	95	5	7	13	42	140	135	2.9	3.5
82	M	87	3.3	2.1	3	N	132	78	124	86	94	98	18	16	95	93	14	22	32	22	131	128	4.4	4.1
83	BK	181	5	3.7	3	N	102	64	115	76	102	94	18	16	93	92	11	8	45	42	136	133	5.7	4.8

S.NO	Name	AST DAY 0	AST DAY 7	ALT DAY 0	ALT DAY 7	ALP DAY 0	ALP DAY 7	Bilirubin DAY 0	Bilirubin DAY 7	Total protein DAY 0	Total protein DAY 7	Albumin DAY 0	Albumin DAY 7	Calcium DAY 0	Parathyroid (if done)	Urine R/E	UPCr(IF DONE)	Urine culture	Blood culture	Any other culture	NEUROLOGICAL FEATURES yes/no	GCS DAY 0	GCS DAY 7	Scenario 1- pt is sedated	RAAS score DAY 0
48	BK	46		35		302		0.8		6		3.3		8.8		PUS CELLS		N	N		Y	9		N	
49	HS	85	88	91	97	139	141	0.3	0.4	11	9	6.2	5	9.2		NORMAL					N	15	15	N	
50	AS	78	64	98	112	202	141	0.8	0.4	7.2	6.4	2.3	2	9		PROTEINURIA		N	N		Y	11	14	N	
51	BS	30	28	30	32	112	141	0.4	0.3	6.9	5.8	3.9	3.7	9	43	PROTEINURIA		N	KLEBSIELLA		N	15	15	N	
52	SK	165	229	165	78	73	98	9	7	5.8	4.9	3.6	2.8	8.4		RBC		N	N		N	15	15	N	
53	NS	52	60	49	54	113	126	0.8	0.7	6.8	6.9	3.4	3.2	8.7		NORMAL					Y	10	12	N	
54	KJ	25	28	14	16	78	84	0.4	1.1	6.2	6.9	3.7	3.2	10		PUS CELLS		N	N		N	15	15	N	
55	GS	28	32	27	40	193	186	1.6	0.9	8.2	7	3.9	3.1	9		PUS CELLS		N			N	15	15	N	
56	GS	58		38		149		6		5.5		1.9		8.2		NORMAL		N	N		Y	14		N	
57	RK	14	16	14	18	75	84	0.6	0.4	7.5	6.8	4.7	4.2	8		NORMAL					N	15	14	N	
58	B	61	84	17	18	106	124	1.05	2.2	6.4	5.8	2.3	2.2	12.5		NORMAL		N	N		Y	12	14	N	
59	VKK	23	24	26	22	86	74	0.9	0.8	6.8	5.6	2.8	2.2	9		NORMAL					N	15	15	N	
60	BS	172	54	92	46	81	70	0.5	0.1	5.8	6	2.9	2.4	8.8		PROTEINURIA	0.7				N	15	15	N	
61	PKJ	19	17	13	12	119	112	0.7	0.5	7	6	3.1	2.8	8.3		NORMAL					N	15	15	N	
62	PS	74	78	172	184	455	436	5.3	3.1	7.2	6.8	2.6	3.2	14		NORMAL		N	N		Y	12	15	N	
63	N	32	22	10	14	160	144	0.3	0.2	4.2	4.4	3.6	3.3	14		NORMAL		N	N		Y	9	15	N	
64	OP	27	22	20	18	142	132	0.2	0.3	6	6.2	2.6	3.2	8.7		PROTEINURIA					N	15	14	N	
65	UK	52	48	29	32	215	200	1.22	0.6	4.8	5.2	2.2	2.4	9		NORMAL					N	15	15	N	
66	HS	20	18	14	12	92	64	0.9	0.8	7.5	7.2	4.2	4	9		NORMAL					N	15	15	N	
67	SS	24	20	16	13	130	128	6.1	5.8	6.1	5.5	2.4	2.2	9		PUS CELLS		N	N		N	15	15	N	
68	N	28	32	20	18	64	74	0.2	1.1	7.2	6.8	4.1	3.8	9.2		PUS CELLS	0.4	E COLI	N		N	15	15	N	
69	RA	31	22	19	17	84	78	0.9	0.7	7.2	6.4	2.3	3.3	8.1		PROTEINURIA		N	N		Y	10	11	N	
70	RS	66	42	81	64	112	57	0.07	0.1	8	7.4	2.7	3.2	9		NORMAL					N	15	15	N	
71	TR	28	26	29	27	124	102	0.6	0.4	8	7	3.5	3.2	10.2		PROTEINURIA					N	15	15	N	
72	NR	17		42		141		3.8		6.3		2.8		7		PROTEINURIA		N	N		Y	10		N	
73	N	13		10		118		0.3		7		3.6		15		RBC PROTEINURIA		N	N		Y	10		N	
74	SK	104		28		112		1		7.2		2.4		9		PUS CELLS		N	N		Y	12		N	
75	KS	101	88	33	38	329	358	5	3	6	2	2.03	2.01	8.9		NORMAL					Y	11	3	Y	0
76	B	35	42	6	12	90	88	0.58	0.4	6.1	5.8	3.3	2.8	9.1		NORMAL					N	15	15	N	
77	ASN	47	22	36	28	112	132	0.2	0.4	6	5.4	2.8	2.7	8.8		NORMAL					N	15	15	N	
78	BS	78	88	52	64	264	223	1	0.9	6.4	5.8	2.4	2.2	9.1		NORMAL					N	15	15	N	
79	SG	85	100	112	141	142	255	0.9	1	6.7	5.5	3.8	3.2	9.9		PUS CELLS		N	N		Y	6	7	Y	-2
80	AS	40	30	25	32	193	178	1.4	0.8	5.3	5	2.7	2.4	9		NORMAL					N	15	15	N	
81	SR	9	12	21	32	65	54	0.5	0.4	5.4	5.5	3.06	8.8	9		PROTEINURIA					N	15	15	N	
82	M	165	102	98	84	112	145	1	1.5	6.4	6.1	2.4	2.3	9		RBC	1.4	N	N		Y	9		N	
83	BK	71	68	67	64	58	54	0.4	0.2	6.6	5.4	2.8	2.4	8.8		NORMAL					N	15	15	N	

S.NO	Name	RAAS score DAY 7	PATIENT IS DELIRIOUS	CAM ICU DAY 0	CAM ICU DAY 7	MMSE DAY 0	MMSE DAY 7	SERIAL 7_DAY 0	SERIAL 7 _DAY 7	MYCOLONUS DAY 0	MYCOLONUS DAY 7	DELUSION/HALLUCINATION DAY 0	DELUSION/HALLUCINATION DAY 7	Asterixis DAY 0	Asterixis DAY 7	Seizure DAY 0	Seizure DAY 7	Sleep inversion DAY 0	Sleep inversion DAY 7	aphasia DAY 0	aphasia DAY 7	cranial nerve involved	MUSCULAR DISORDERSDAY 0	MUSCULAR DISORDERS DAY 7	Tremor/involuntary movements DAY 0
48	BK		Y	Y		0		0		N		Y		N		N		Y		N		N	N		N
49	HS		N	N	N	28	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
50	AS		Y	Y	N	0	20	4	5	N	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	Y
51	BS		N	N	N	26	28	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
52	SK		N	N	N	27	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
53	NS		Y	Y	N	0		0	1	Y	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	N
54	KJ		N	N	N	27	29	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
55	GS		N	N	N	28	29	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
56	GS		N	N		15		1		N		N		N		Y		N		N		N	N		N
57	RK		N	N	N	26	25	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
58	B		Y	Y	N	0	8	0	2	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
59	VKK		N	N	N	27	26	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
60	BS		N	N	N	26	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
61	PKJ		N	N	N	26	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
62	PS		Y	Y	N	0	10	1	2	N	N	Y	N	N	N	Y	N	Y	Y	N	N	N	N	N	N
63	N		Y	Y	N	0	18	0	2	N	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	Y
64	OP		N	N	N	28	28	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
65	UK		N	N	N	27	28	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
66	HS		N	N	N	26	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
67	SS		N	N	N	29	28	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
68	N		N	N	N	27	28	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
69	RA		Y	Y	N	0	0	0	1	N	N	N	N	Y	Y	N	N	Y	Y	N	N	N	N	N	N
70	RS		N	N	N	26	27	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
71	TR		N	N	N	26	26	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
72	NR		Y	Y		0		0		N		N		N		N		N		N		N	N		N
73	N		Y	Y		0		0		N		Y		N		Y		N		N		N	N		N
74	SK		Y	Y		0		0		Y		N		N		N		N		N		N	N		N
75	KS	-3	N	N	N	0		1	0	N	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	N
76	B		N	N	N	26	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
77	ASN		N	N	N	27	26	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
78	BS		N	N	N	27	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
79	SG	-1	Y	Y	Y	0	0	0	0	Y	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N
80	AS		N	N	N	28	28	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
81	SR		N	N	N	26	27	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
82	M		Y	Y				1	3	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
83	BK		N	N	N	27	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N

S.NO	Name	Tremor/involuntary movements DAY 7	Wasting DAY 0	Wasting DAY 7	Fasciculation DAY 0	Fasciculation DAY 7	SENSORY SYSTEM	ABNORMAL DEEP TENDON reflexes	LOSS OF URINARY CONTROL DAY 0	LOSS OF URINARY CONTROL DAY 7	Night diarrhea DAY 0	Night diarrhea DAY 7	Neck rigidity DAY 0	Neck rigidity DAY 7	ADDITIONAL INVESTIGATIONS AS AND WHEN INDICATED_CT HEAD	MRI	EEG	CSF ANALYSIS-as and when required	USG abdomen
48	BK		N		N		N	N	N		N		N		X	diffuse oedema in bilateral cerebral hemispheres and hyperintensity in bilateral basal ganglia and thalamus	X	NORMAL	FATTY LIVER ASCITES
49	HS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	DILATED BOWEL LOOPS
50	AS	N	N	N	N	N	N	N	N	N	N	N	N	N	NORMAL	PRES	X	X	INTESTINAL OBSTRUCTION
51	BS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
52	SK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ASCITES PLEURAL EFFUSION
53	NS	N	N	N	N	N	N	N	N	N	N	N	N	N	NORMAL	X	X	X	HEPATOMEGALY
54	KJ	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PERINEPHRIC STRANDING
55	GS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CYSTITIS
56	GS		N		N		N	N	N		N		N		HYPODENSITIES B/L PERIVENTRICULAR WHITE MATTER	X	X	X	CLD
57	RK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ACUTE CHOLECYSTITIS
58	B	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ASCITES
59	VKK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
60	BS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
61	PKJ	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CHOLELITHIASIS
62	PS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HYDRONEPHROSIS
63	N	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
64	OP	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
65	UK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CHOLELITHIASIS
66	HS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
67	SS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CYSTITIS
68	N	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PERINPEHRIC STARNDING
69	RA	N	N	N	N	N	X	N	N	N	N	N	N	N	X	X	X	X	CLD
70	RS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
71	TR	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB EDEMA
72	NR		N		N		X	N	N		N		Y		NORMAL	X	X	WITHINNORMAL LIMIT	HEPATOSPELNOMEGALY
73	N		N		N		X	N	N		N		N		X	MILD SMOOTH DURAL THICKENING	X	ELEVATED PROTEIN, NEUTROPHILIC PREDOMINANT	SPLENOMEGALY
74	SK		N		N		X	N	N		N		Y		CEREBRAL EDEMA	X	X	X	FATTY LIVER
75	KS	N	Y	Y	N	N	X	N	N	N	N	N	Y	N	VENTRICULOMEGALY,ATROPHY	X	X	HIGH ADA, HIGH PROTEIN MNC	FATTY LIVER RENAL CHANGES
76	B	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
77	ASN	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB EDEMA
78	BS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	SPLENOMEGALY
79	SG	N	N	N	N	N	X	N	N	N	N	N	N	N	NORMAL	FOCALINCREASET2OLIVARYNUCLEUS	X	WITHINNORMAL LIMIT	CHOLELITHIASIS
80	AS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	B/L BULKY KIDNEY
81	SR	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIIVER, ASCITES LYMPH NODES
82	M	N	N	N	N	N	X	N	N	N	N	N	N	N	NORMAL	NORMAL	X	X	SPLENOMEGALY
83	BK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CHOLECYSTITIS

S.NO	Name	Age	Sex	Presenting complaints	ICU STAY	hypertension	diabetes mellitus 2	CLD	CAD	Any nephrotoxic drugs prior to admission	Type of nephrotoxic drugs	H/O Alcohol/smoking/substance abuse	Mechanical ventilatory support	Ionotropic support	qSOFA _Day 0	qSOFA _Day 7	CAUSE	AKI etiology	sepsis present	septic shock present	source of sepsis	UREA DAY 0
84	SCS	60	M	WOUND FOOT	N	Y	Y	Y	N	N		ALCOHOL	N	N	0	0	INFECTION	DIABETIC FOOT	N	N		112
85	CK	66	F	ABNORMAL BODY MOVEMENTS	Y	N	N	N	N	N		N	Y	Y	2	1	INFECTION	PNEUMONIA	Y	N	RESPIRATORY	61
86	AK	37	F	BURNING MICTURITION	N	N	N	N	N	N		N	N	N	0	0	INFECTION	UTI	N	N		173
87	JS	65	M	YELLOWING OF SCLERA	N	Y	N	N	N	N		N	N	N	0	0	TROPICAL FEVER	LEPTOSPIRA	N	N		103
88	MS	34	F	LOOSE STOOLS	N	N	N	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		33
89	DS	39	F	LOOSE STOOLS	Y	N	N	Y	N	N		N	N	N	2	2	INFECTION	ACUTE GASTROENTERITIS	N	N		34
90	SS	70	M	ALTERED SENSORIUM, FEVER	Y	N	N	N	N	N		OPIOD	Y	Y	3	3	INFECTION	HYPOVOLUMEA	N	N		83
91	M	72	M	BURNING MICTURITION	N	Y	Y	N	Y	N		N	N	N	0	0	INFECTION	UTI	N	N		246
92	D	30	M	MALAISE	N	N	N	N	N	N		N	N	N	0	0	MISC	AML	N	N		42
93	J	40	M	BURNING MICTURITION	N	N	Y	N	N	N		N	N	N	0	0	INFECTION	UTI	N	N		41
94	N	62	F	ABDOMINAL DISTENSION	N	Y	Y	N	Y	N		N	N	N	0	0	INFECTION	UTI	N	N		84
95	KR	53	M	FEVER PAIN ABDOMEN	N	Y	Y	Y	N	N		N	N	N	2	0	INFECTION	ACUTE GASTROENTERITIS	N	N		55
96	SS	70	M	ALTERED SENSORIUM	Y	Y	N	N	N	N		N	Y	Y	2	2	INFECTION	TUBERCULOSIS	N	N		83
97	MS	70	M	LOOSE STOOLS	N	Y	N	N	N	N		N	N	N	0	0	MISC	IgG4 DISEASE	N	N		63
98	GS	35	M	PAIN ABDOMEN	Y	N	N	N	N	N		ALCOHOL	Y	Y	2	2	PANCREATITIS	ACUTE PANCREATITIS	N	N		144
99	U	65	F	FEVER , RASH	Y	Y	N	N	N	N		N	N	N	0	0	INFECTION	VIRAL FEVER	N	N		174
100	S	21	F	LOOSE STOOLS	N	N	N	N	N	N		N	N	N	0	0	TROPICAL FEVER	DENGUE	N	N		34
101	SK	56	F	ABNORMAL BODY MOVEMENTS	N	Y	N	N	N	N		N	N	Y	2	0	TROPICAL FEVER	COMPLICATED TROPICAL FEVER	Y	Y	TROPICAL FEVER	194
102	RS	43	M	ALTERED SENSORIUM	Y	N	N	N	N	N		N	N	N	2	2	MISC	HYPERCALCEMIA	N	N		99
103	AB	33	M	WEAKNESS HEADACHE	N	N	N	N	N	N		N	N	N	0	0	INFECTION	UTI	N	N		68
104	VKY	36	M	PAIN ABDOMEN	N	N	N	N	N	N		N	N	N	0	0	SURGICAL	INTESTINAL OBTSRUCTION	N	N		67
105	SS	60	F	BURNING MICTURITION	N	N	Y	N	N	N		N	N	N	0	0	TROPICAL FEVER	DENGUE	N	N		47
106	HS	57	M	MALAISE	N	N	N	N	N	N		ALCOHOL	N	N	1	0	TROPICAL FEVER	DENGUE	N	N		185
107	SK	76	F	BURNING MICTURITION	N	Y	Y	Y	N	N		N	N	N	0	0	INFECTION	UTI	N	N		106
108	KL	86	M	DYSPNEA	N	Y	Y	N	Y	N		N	N	Y	1	1	CARDIORENAL	CARDIORENAL	N	N		89
109	KL	60	M	DECREASED URINE OUTPUT	Y	N	Y	Y	N	N		N	N	Y	1	0	SURGICAL	INTESTINAL OBTSRUCTION	N	N		94
110	KR	49	F	PAIN ABDOMEN	Y	N	Y	N	Y	N		N	N	N	0	0	INFECTION	ENTERIC FEVER	N	N		48
111	S	40	M	PAIN ABDOMEN	N	N	Y	N	N	N		N	N	N	0	0	INFECTION	PYELONEPHRITIS	N	N		90
112	KK	58	F	DYSPNEA COUGH	N	N	N	N	N	N		N	N	N	0	0	INFECTION	PNEUMONIA	N	N		59
113	SD	79	F	CONSTIPATION	Y	Y	N	N	N	N		N	N	N	0	0	SURGICAL	INTESTINAL OBTSRUCTION	N	N		194
114	CSD	74	M	DECREASED AROUSAL	Y	Y	Y	N	Y	N		N	N	Y	1	0	MISC	DIABETIC KETOACIDOSIS	N	N		66
115	S	24	F	LOOSE STOOLS	N	N	N	N	N	N		N	N	N	0	0	INFECTION	UTI	N	N		57
116	GKJ	20	M	HEMATURIA	Y	N	N	N	N	N		N	N	Y	2	1	INFECTION	UTI	Y	Y	UROSEPSIS	140
117	CK	65	F	DYSPNEA	Y	Y	Y	N	N	N		N	N	Y	3	2	INFECTION	PNEUMONIA	Y	Y	RESPIRATORY	111
118	RL	60	M	PAIN ABDOMEN	N	N	Y	N	Y	N		Y	N	N	1	0	CARDIORENAL	CARDIORENAL	N	N		90
119	MS	60	F	YELLOWING OF SCLERA	N	N	N	N	Y	N		N	N	N	0	0	TROPICAL FEVER	LEPTOSPIRA	N	N		95
120	KK	62	F	DECREASED APPETITE	N	N	N	Y	N	N		ALCOHOL	N	N	0	0	TROPICAL FEVER	DENGUE	N	N		86
121	SS	49	F	BURNING MICTURITION	N	N	Y	N	N	N		N	N	N	0	0	INFECTION	UTI	N	N		115
122	AK	42	M	DECREASED URINE OUTPUT	Y	N	N	Y	N	N		N	N	Y	2	1	HRS	HRS	N	N		89
123	PS	63	M	DYSPNEA	N	N	N	N	N	N		N	N	N	1	0	INFECTION	PNEUMONIA	N	N		48
124	MS	61	F	MALAISE	N	N	N	N	N	N		N	N	N	0	0	CARDIORENAL	CARDIORENAL	N	N		84
125	SR	63	F	MALAISE	N	Y	N	N	N	N		N	N	N	0	0	INFECTION	UTI	N	N		265

S.NO	Name	UREa day 7	Serum creatinine	CREAT DAY 7	AKI STAGE	Dialysis session	SBP_DAY 0	DBP_DAY 0	SBP_DAY 7	DBP_DAY 7	Heart rate DAY 0	Heart rate DAY 7	Respiratory rate DAY 0	Respiratory rate DAY 7	Saturation oxygen DAY 0	Saturation oxygen DAY 7	TLC DAY 0	TLC DAY 7	Platelet count DAY 0	Platelet count DAY 7	Sodium DAY 0	Sodium DAY 7	Potassium DAY 0	Potassium DAY 7
84	SCS	42	4.9	1.8	3	N	110	88	132	86	122	88	18	16	95	97	18	11	140	112	131	128	5.2	4.8
85	CK	164	1.8	2.8	1	Y	150	115	115	78	97	84	18	16	95	95	27	17	180	175	139	131	2.4	3.2
86	AK	70	3.1	1.7	2	N	128	84	122	86	94	88	14	16	97	96	14	11	348	252	138	148	5.2	4.8
87	JS	74	1.7	0.8	1	N	112	78	120	84	94	97	16	14	97	96	9	7	142	110	139	137	3.8	4.1
88	MS	64	1.6	1.8	1	N	122	85	118	76	97	98	16	14	97	99	15	11	186	212	141	137	4.1	4.6
89	DS	32	2.6	3.18	2	N	94	64	110	74	98	94	16	14	94	93	11	34	489	539	135	138	5.5	4.2
90	SS	112	1.7	1.9	1	N	132	84	128	76	95	92	20	18	94	96	31	21	292	258	131	137	5	4.5
91	M	154	11	6	3	Y	102	74	115	72	86	84	18	16	93	91	14	8	114	154	141	142	3.8	4.5
92	D	55	2.1	1.2	1	N	132	78	128	74	85	88	14	16	96	94	29	54	13	10	143	136	4.6	4.1
93	J	22	1.6	0.9	1	N	112	68	124	82	76	74	14	14	97	98	21	14	383	324	131	128	4.8	4.4
94	N	65	1.7	0.9	1	N	144	78	132	84	68	72	18	16	96	94	22	9	199	145	126	128	5.3	4.8
95	KR	59	2.8	3.09	2	N	128	86	122	78	86	88	20	18	97	96	5	4.8	90	74	115	128	3.1	3.8
96	SS	112	1.7	1.9	1	N	84	54	98	66	100	98	18	14	88	95	24	14	364	345	130	133	6.3	5.1
97	MS	34	2	0.9	1	N	110	72	132	86	94	84	18	16	95	93	4.8	3.1	154	110	139	128	4.8	4.5
98	GS	171	8	9	3	Y	86	54	111	70	112	102	26		82	92	11	15	78	98	127	133	4.9	5
99	U	102	1.6	0.7	1	N	132	88	112	76	98	76	16	18	95	94	7	10	487	455	116	120	5.2	4.8
100	S	61	2.1	1	1	N	122	74	126	68	84	82	14	16	99	98	20	14	290	248	121	134	4.4	3.8
101	SK	122	6	3.1	3	Y	78	55	122	86	92	84	16	14	97	94	7	6.4	40	50	133	128	4.4	4.1
102	RS	42	1.8	1.15	1	N	112	88	114	76	99	105	16	14	93	97	2.6	2	179	290	130	131	3.4	5.2
103	AB	28	1.9	1	1	N	112	84	132	87	98	85	16	14	94	98	12	10	268	245	134	128	4.5	4.2
104	VKY	111	2.3	3.9	2	N	132	84	124	76	72	84	14	16	95	94	29	12	150	170	136	138	4.07	3.9
105	SS	41	1.8	1.4	1	N	122	78	132	84	74	98	14	14	98	97	14	8	354	322	134	135	4.5	5.01
106	HS	156	5.5	2.3	3	N	100	78	122	87	89	87	16	14	94	96	6	7	45	40	137	140	4.14	3.69
107	SK	132	1.7	1.2	1	N	116	84	132	88	86	87	16	14	94	95	14	10	183	164	135	133	4.3	3.8
108	KL	78	1.8	1.6	1	N	111	82	132	92	86	88	20	18	94	96	11	9	201	200	136	141	4	5
109	KL	40	2.7	1.18	2	N	100	78	98	69	99	72	18	16	95	96	21	9	91	76	138	135	4.1	3.9
110	KR	52	2.3	1.2	2	N	100	72	112	89	74	72	14	16	90	98	44	48	150	64	138	144	4.4	3.8
111	S	82	3.5	2.4	3	N	122	78	112	84	86	84	20	18	94	95	11	8	142	156	133	136	4.14	4.07
112	KK	45	2	1.01	1	N	144	80	138	84	84	88	14	16	97	96	25	12	641	457	131	136	3.6	3.8
113	SD	46	2.4	0.9	2	N	132	99	141	88	86	89	20	18	94	95	13	18	130	252	145	132	4.03	4.3
114	CSD	70	1.7	1.6	1	N	112	75	132	88	115	100	22	14	92	94	21	9	91	245	134	140	4.7	4.5
115	S	43	1.6	0.8	1	N	100	70	110	80	122	88	20	16	95	96	18	9	305	220	131	133	4.1	3.7
116	GKJ	89	7.2	5.7	3	Y	94	68	102	88	98	84	22	18	88	94	38	14	445	422	142	142	4.5	3.5
117	CK	79	2.6	1.6	2	N	91	62	94	68	122	100	20	24	87	84	22	24	188	150	131	137	5	5.7
118	RL	108	4.16	4.84	3	N	112	78	122	84	112	92	18	18	92	94	15	11	120	118	129	141	6.7	5.1
119	MS	97	1.8	1.6	1	N	122	84	114	78	102	94	16	14	95	94	10	17	80	66	136	137	5.9	6
120	KK	70	1.6	1.06	1	N	110	78	132	86	72	84	14	16	93	94	8	10	140	110	147	156	3.5	3.6
121	SS	73	5	4.6	3	N	133	78	128	84	74	78	14	16	95	96	31	15	120	130	136	137	6.2	5.1
122	AK	94	3.14	2.5	3	Y	88	64	110	78	86	84	20	18	88	92	6	6.5	6	13	140	134	3.5	3.7
123	PS	14	1.6	0.7	1	N	138	70	121	74	94	86	20	14	90	94	20	18	222	186	136	137	4.4	3.04
124	MS	60	1.7	0.9	1	N	110	82	128	76	102	78	20	18	95	92	6	5	136	150	142	141	2.9	4.1
125	SR	162	7.5	3.9	3	Y	150	88	132	80	74	78	14	16	95	94	8.1	6	196	138	129	135	4.8	4.7

S.NO	Name	AST DAY 0	AST DAY 7	ALT DAY 0	ALT DAY 7	ALP DAY 0	ALP DAY 7	Bilirubin DAY 0	Bilirubin DAY 7	Total protein DAY 0	Total protein DAY 7	Albumin DAY 0	Albumin DAY 7	Calcium DAY 0	Parathyroid (if done)	Urine R/E	UPCr(IF DONE)	Urine culture	Blood culture	Any other culture	NEUROLOGICAL FEATURES yes/no	GCS DAY 0	GCS DAY 7	Scenario 1- pt is sedated	RAAS score DAY 0
84	SCS	130	112	88	78	252	202	2.2	0.9	6.9	5.5	3.2	2.7	8.6		PROTEINURIA		N	N	ASCITIC-PSEUDOMONAS	N	15	15	N	
85	CK	29	32	28	34	69	87	3.5	1.2	7.5	6.5	3.3	2.8	8.8		PUS CELLS, PROTEINS		N	N		Y	6	8	Y	-2
86	AK	36	32	25	20	221	200	0.46	0.4	7.4	6.5	3.4	2.8	8.9		PUS CELLS		N	N		N	15	15	N	
87	JS	55	46	55	58	188	242	2.2	3.2	5.6	5.9	2.7	2.8	8.7		NORMAL					N	15	15	N	
88	MS	36	28	19	16	183	112	0.6	0.4	5	6	1.2	2.2	9		NORMAL					N	15	15	N	
89	DS	106	114	180	178	312	245	1.1	2	4.7	4.8	2.01	2	8		PUS CELLS			N		N	15	14	N	
90	SS	99	98	25	26	151	160	6.2	8.4	7.5	8.2	2.4	2.2	10.1		RBC		N	N		Y	8	10	N	
91	M	74	82	78	68	112	116	0.9	1.1	6.4	7	2.3	2.1	9.8		PUS CELLS		N	N		N	15	15	N	
92	D	120	114	81	76	167	172	0.8	0.6	7.3	7.1	4.1	3.8	10		NORMAL		N	N		N	15	15	N	
93	J	29	28	32	31	236	201	0.7	0.4	7.2	7.1	3.5	3.4	9.4		PUS CELLS		N	N		N	15	15	N	
94	N	4	10	6	12	130	122	0.5	0.4	4.9	5.1	2.4	2.2	8		PROTEINURIA					N	15	15	N	
95	KR	124	136	36	38	150	164	27	32	5.8	4.5	2.1	1.9	8		NORMAL			N		Y	10	14	N	
96	SS	181	220	66	76	208	320	6.2	8.4	5.5	6	2.5	2.6	10		NORMAL			N		Y	8	9	Y	1
97	MS	24	20	21	18	16	14	0.17	0.16	6.6	5.9	3.1	2.8	10.1		PUS CELLS, RBC					N	15	15	N	
98	GS	63	30	90	38	67	62	1.1	0.6	5	4.7	2.3	2.01	8.8		PROTEINURIA			N		Y	13	9	Y	
99	U	21	24	11	14	166	154	1.1	0.9	8.3	7	3.8	3.2	8.8		PUS CELLS					Y	14	15	N	
100	S	33	24	28	16	121	92	0.4	0.2	11	8	5.5	4	12.5		NORMAL					N	15	15	N	
101	SK	170	154	83	80	138	122	15	12	5.7	5.2	3.2	2.8	8.4		RBC PROTEINURIA					Y	10	12	N	
102	RS	25	28	14	16	255	324	1	1.6	5.2	4.8	2.4	2.7	14	1.08	PROTEINURIA			N		Y	10	11	N	
103	AB	14	22	48	35	142	101	0.36	0.2	7	6	2.5	3.2	8		NORMAL	0.51	N	N		N	15	15	N	
104	VKY	60	45	85	82	92	88	0.4	0.2	10	8	6.1	5.4	10.2		NORMAL					N	15	15	N	
105	SS	20	18	16	14	139	141	0.5	0.3	6.7	6.4	3.09	2.8	8.4		PUS CELLS	1.4	N	N		N	15	15	N	
106	HS	23	26	49	48	50	70	0.8	0.7	7	6.5	3.9	3.2	9		PROTEINURIA					N	15	15	N	
107	SK	278	242	204	120	65	74	0.59	1	6	5.8	3.48	3.2	9		PUS CELLS		N	N		N	15	15	N	
108	KL	20	18	55	54	135	130	0.9	1	6.5	6.2	2.8	2.9	8.9		PROTEINURIA	0.4				N	15	15	N	
109	KL	54	52	40	38	73	78	1.07	0.9	6.4	6.3	2.9	2.8	9		PROTEINURIA	0.3				N	15	15	N	
110	KR	284	228	90	82	205	150	1.2	1.9	4.6	5.2	2.4	2.9	11		PUS CELLS	1				Y	10	13	N	
111	S	59	54	63	64	167	158	0.2	0.1	5.3	6	2.6	2.4	10		PROTEINURIA, PUS CELLS	0.77	N	N		N	15	15	N	
112	KK	31	27	21	19	168	131	0.3	0.36	6.7	5.8	2.5	2.4	8.5		PUS CELLS		KLEBSIELLA	N		N	15	15	N	
113	SD	99	76	85	84	118	114	2.5	2	5	4.8	2.2	1.9	8.1		PROTEINURIA		N	N		N	15	14	N	
114	CSD	19	17	18	20	78	77	0.2	0.3	5.9	5.7	3.6	3.1	7		GLUCOSE					Y	10	15	N	
115	S	13	14	17	16	143	132	0.8	0.7	5.5	5.4	2.6	2.9	8.8		NORMAL					N	15	15	N	
116	GKJ	621	600	850	450	97	92	1.6	2.2	9.7	7.4	5.3	4.8	8		PUS CELLS	95				N	15	15	N	
117	CK	14	16	18	20	109	115	0.29	0.1	6	7	3.2	3.1	10		NORMAL		CANDIDA STELLOIDES	N	SPUTUM-ENTEROCOCCUS	N	15	15	N	
118	RL	152	140	132	122	56	48	1.2	0.9	6.3	5.8	3.7	2.8	10		NORMAL					N	15	15	N	
119	MS	26	24	69	74	30	42	9	10	6.9	7.2	3.2	4.1	9		RBC					N	15	15	N	
120	KK	59	90	52	62	113	104	1.7	2.3	5.5	5	2.7	2.6	10		NORMAL					N	15	15	N	
121	SS	61	58	36	42	167	145	5.6	4	6.2	5.8	2.4	2.3	9.2		NORMAL		N	N		N	15	15	N	
122	AK	83	74	26	32	95	105	1.9	1	5.8	6.1	2.7	2.6	9.8		NORMAL		N	N		N	15	15	N	
123	PS	53	76	52	94	88	60	0.14	0.84	6.2	5.8	3.5	3.2	10		NORMAL					N	15	15	N	
124	MS	33	28	37	26	280	245	0.4	0.3	5.3	6	2.7	3.2	10		NORMAL					N	15	15	N	
125	SR	172	168	121	118	85	87	0.8	0.6	7.7	7.1	3.4	3.2	9.1		YEAST, PUS CELLS		N	N		N	15	15	N	

S.NO	Name	RAAS score DAY 7	PATIENT IS DELIRIOUS	CAM ICU DAY 0	CAM ICU DAY 7	MMSE DAY 0	MMSE DAY 7	SERIAL 7_DAY 0	SERIAL 7 _DAY 7	MYCOLONUS DAY 0	MYCOLONUS DAY 7	DELUSION/HALLUCINATION DAY 0	DELUSION/HALLUCINATION DAY 7	Asterixis DAY 0	Asterixis DAY 7	Seizure DAY 0	Seizure DAY 7	Sleep inversion DAY 0	Sleep inversion DAY 7	aphasia DAY 0	aphasia DAY 7	cranial nerve involved	MUSCULAR DISORDERSDAY 0	MUSCULAR DISORDERS DAY 7	Tremor/involuntary movements DAY 0
84	SCS		N	N	N	27	28	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
85	CK	-1	N	N	N			0	0	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
86	AK		N	N	N	26	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
87	JS		N	N	N	24	25	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
88	MS		N	N	N	27	28	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
89	DS		N	N	N	24	25	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
90	SS		Y	Y	Y	0	0	0	0	N	N	N	N	Y	Y	N	N	Y	Y	N	N	N	N	N	Y
91	M		N	N	N	27	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
92	D		N	N	N	28	29	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
93	J		N	N	N	26	27	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
94	N		N	N	N	25	25	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
95	KR		N	N	N	0	10	0	2	N	N	N	N	N	N	Y	N	Y	Y	N	N	N	N	N	N
96	SS	-2	N	N	N	0	0	0	0	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y
97	MS		N	N	N	27	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
98	GS	-1	N	N	N	4	0	2	0	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y
99	U		N	N	N	26	27	3	4	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N
100	S		N	N	N	26	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
101	SK		Y	Y	Y	0	0	0	0	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N
102	RS		Y	Y	Y	0	0	0	2	Y	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	Y
103	AB		N	N	N	26	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
104	VKY		N	N	N	26	26	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
105	SS		N	N	N	27	28	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
106	HS		N	N	N	27	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
107	SK		N	N	N	26	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
108	KL		N	N	N	28	27	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
109	KL		N	N	N	28	29	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
110	KR		N	N	N	0	0	0	0	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
111	S		N	N	N	27	28	5	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
112	KK		N	N	N	27	28	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
113	SD		N	N	N	29	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
114	CSD		N	N	N	0	15	0	2	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N
115	S		N	N	N	29	30	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
116	GKJ		N	N	N	26	26	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
117	CK		N	N	N	22	24	3	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
118	RL		N	N	N	28	29	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
119	MS		N	N	N	27	28	5	5	N	N	N	N	N	N	N	N	N	N	N		N	N		N
120	KK		N	N	N	28	29	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
121	SS		N	N	N	26	28	5	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
122	AK		N	N	N	25	26	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
123	PS		N	N	N	28	29	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
124	MS		N	N	N	28	27	5	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
125	SR		N	N	N	28	27	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N

S.NO	Name	Tremor/involuntary movements DAY 7	Wasting DAY 0	Wasting DAY 7	Fasciculation DAY 0	Fasciculation DAY 7	SENSORY SYSTEM	ABNORMAL DEEP TENDON reflexes	LOSS OF URINARY CONTROL DAY 0	LOSS OF URINARY CONTROL DAY 7	Night diarrhea DAY 0	Night diarrhea DAY 7	Neck rigidity DAY 0	Neck rigidity DAY 7	ADDITIONAL INVESTIGATIONS AS AND WHEN INDICATED_CT HEAD	MRI	EEG	CSF ANALYSIS-as and when required	USG abdomen
84	SCS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CLD
85	CK	N	Y	Y	N	N	X	N	N	N	N	N	N	N	NORMAL	EMBOLICINFARCT, HYPERINTENSE CEREBELLAR HEMISPHERES	X	X	PERINEPHRIC STRANDING
86	AK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HYDRONEPHROSIS
87	JS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB EDEMA
88	MS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
89	DS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CLD
90	SS	Y	N	N	N	N	X	N	N	N	N	N	N	N	NORMAL	MASTOIDITIS,CHRONICATOPHY	DIFFUSEENCEPAHLOPATHY		RENAL CHANGES
91	M	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HYDRONEPHROSIS
92	D	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ACUTE RENAL DISEASE
93	J	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CYSTITIS
94	N	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
95	KR	N	N	N	N	N	X	N	N	N	N	N	N	N	X	X	X	X	CLD
96	SS	N	N	N	N	N	X	N	N	N	N	N	Y	N	X	PACHYMENINGITIS,SUBDURALEMPYEMA,VENTRICULITIS,HYDROCEPHALUS	X	X	SPLENOMEGALY
97	MS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	RENAL CHANGES
98	GS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PANCREATITIS
99	U	N	N	N	N	N	N	N	N	N	N	N	N	N	NCC	X	X	X	ASCITES
100	S	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	SPLENOMEGALY
101	SK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB SLUDGE
102	RS	Y	N	N	N	N	X	N	N	N	N	N	N	N	X	NORMAL	X	NORMAL	HEPATOSPELNOMEGALY
103	AB	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PERINEPHRIC STRANDING
104	VKY	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
105	SS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PERINEPHRIC STRANDING
106	HS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB SLUDGE
107	SK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	SPLENOMEGALY
108	KL	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
109	KL	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CHOLELITHIASIS
110	KR	N	N	N	N	N	X	N	N	N	N	N	Y	Y	NORMAL	X	X	X	GB EDEMA
111	S	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HYDRONEPHROSIS
112	KK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NEPHROLITHIASIS
113	SD	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	DILATED GUT LOOPS
114	CSD	N	Y	Y	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
115	S	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
116	GKJ	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ASCITES RENAL PARENCHYMAL CHANGES
117	CK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	RENAL CORTICAL CYST
118	RL	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER ASCITES
119	MS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB EDEMA
120	KK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB EDEMA
121	SS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CYSTITIS
122	AK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HEPATOSPLENOMEGALY
123	PS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ASCITES
124	MS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER, CHOLELITHIASIS
125	SR	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL

S.NO	Name	Age	Sex	Presenting complaints	ICU STAY	hypertension	diabetes mellitus 2	CLD	CAD	Any nephrotoxic drugs prior to admission	Type of nephrotoxic drugs	H/O Alcohol/smoking/substance abuse	Mechanical ventilatory support	Ionotropic support	qSOFA _Day 0	qSOFA _Day 7	CAUSE	AKI etiology	sepsis present	septic shock present	source of sepsis	UREA DAY 0
126	HK	80	F	ABNORMAL BODY MOVEMENTS	Y	Y	N	N	N	N		N	N	N	1	1	INFECTION	UTI	N	N		73
127	CSG	58	M	MALAISE	N	N	Y	N	Y	N		N	N	N	0	0	MISC	DIABETIC KETOACIDOSIS	N	N		145
128	BS	25	M	BLACK STOOLS	Y	N	N	N	N	N		N	Y	Y	3	2	INFECTION	PNEUMONIA	Y	Y	RESPIRATORY	258
129	SS	36	M	DECREASED URINE OUTPUT	Y	N	N	N	N	Y	PARAQUAT	ALCOHOL	N	N	1	1	NEPHROTOXIN	NEPHROTOXIN	N	N		25
130	KL	60	M	PAIN ABDOMEN	Y	N	N	Y	N	N		ALCOHOL	N	Y	1	0	INFECTION	UTI	N	N		94
131	BB	52	M	DECREASED URINE OUTPUT, ALTERED SENSORIUM	Y	N	N	N	N	N		IVDU	N	Y	3	3	INFECTION	BACTEREMIA	Y	Y	BACTEREMIA	48
132	MK	56	F	PAIN IN EYE	Y	N	N	N	N	N		N	N	Y	1	0	INFECTION	MUCROMYCOSIS	Y	Y	ORBITAL	32
133	MD	50	F	FEVER	N	N	N	N	N	N		N	N	Y	1	0	CARDIORENAL	CARDIORENAL	N	N		220
134	BS	56	F	DYSPNEA	N	N	N	N	N	N		SMOKING	N	Y	1	1	HYPOVOLUMEA	HYPOVOLUMEA	N	N		74
135	SR	62	F	DECREASED URINE OUTPUT	N	N	N	N	N	N		N	N	N	1	0	INFECTION	UTI	N	N		147
136	TS	58	M	WOUND FOOT	N	N	Y	N	N	N		OPIOD, ALCOHOL	N	Y	2	1	INFECTION	DIABETIC FOOT	Y	Y	DIABETIC FOOT	91
137	KK	56	M	FEVER COUGH	N	N	N	N	N	N		N	N	N	0	0	MISC	ANCA VASCULITIS	N	N		42
138	SR	74	M	FEVER	N	N	N	N	N	Y	COLISTIN	N	N	N	0	0	NEPHROTOXIN	NEPHROTOXIN	N	N		64
139	PS	39	M	DECREASED APPETITE	N	N	N	N	N	N		ALCOHOL	N	N	1	0	TROPICAL FEVER	DENGUE	N	N		86
140	MB	47	F	FEVER	N	N	N	N	N	N		N	N	N	2	0	INFECTION	PYELONEPHRITIS	Y	Y	UROSEPSIS	87
141	PK	70	F	ALTERED SENSORIUM	Y	N	N	N	N	Y	AMIKACIN	N	N	N	2	1	MISC	HYPERCALCEMIA	N	N		81
142	KK	58	F	DYSPNEA COUGH	Y	N	N	N	N	N		N	N	N	1	0	INFECTION	PNEUMONIA	N	N		59
143	DS	42	M	CHEST PAIN	N	Y	N	N	Y	N		N	N	N	0	0	CARDIORENAL	CARDIORENAL	N	N		81
144	JK	74	F	DECREASED URINE OUTPUT	N	Y	N	N	N	Y	NSAIDS	N	N	N	1	0	NEPHROTOXIN	NEPHROTOXIN	N	N		132
145	BB	86	M	DYSPNEA COUGH	N	Y	Y	N	Y	N		N	N	N	0	0	INFECTION	PNEUMONIA	N	N		89
146	SK	64	F	PAIN ABDOMEN	N	N	N	N	N	N		N	N	N	1	0	MISC	AML	N	N		56
147	CK	30	F	LOOSE STOOLS	N	N	N	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		52
148	JK	45	F	SNAKE BITE	Y	N	N	N	N	N		N	N	N	1	0	MISC	SNAKE BITE	N	N		100
149	PK	86	F	ALTERED SENSORIUM	Y	Y	N	N	N	Y	VITAMIN D	N	N	N	1	0	MISC	HYPERCALCEMIA	N	N		24
150	HK	65	F	LOOSE STOOLS	N	Y	N	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		45
151	J	20	M	DECREASED URINE OUTPUT	Y	N	N	N	N	Y	PARAQUAT	ALCOHOL	N	N	1	2	NEPHROTOXIN	NEPHROTOXIN	N	N		64
152	H	47	M	LOOSE STOOLS	N	N	Y	N	N	N		NICOTINE	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		26
153	RK	83	M	VOMITING	N	N	N	N	N	N		ALCOHOL	N	Y	2	1	INFECTION	ACUTE GASTROENTERITIS	Y	Y	GASTROENTEROLOGY	82
154	d	84	M	BURNING MICTURITION	N	N	Y	N	N	N		N	N	N	0	0	INFECTION	UTI	N	N		200
155	R	27	F	DECREASED URINE OUTPUT	Y	N	N	N	N	Y	PARAQUAT	N	N	N	1	2	NEPHROTOXIN	NEPHROTOXIN	N	N		57
156	V	24	M	PEDAL EDEMA	N	N	N	N	N	Y	OPIOD	N	N	N	1	1	NEPHROTOXIN	NEPHROTOXIN	N	N		127
157	R	25	M	PAIN ABDOMEN	Y	N	N	N	N	N		ALCOHOL	N	Y	2	3	PANCREATITIS	PANCREATITIS	N	N		98
158	B	64	M	DECREASED URINE OUTPUT	Y	N	Y	N	N	N		OPIOD	N	Y	2	2	INFECTION	PYELONEPHRITIS	Y	Y	UROSEPSIS	138
159	P	50	M	DECREASED URINE OUTPUT	Y	N	Y	N	N	N		ALCOHOL	N	Y	2	1	INFECTION	PYELONEPHRITIS	Y	Y	UROSEPSIS	169
160	BS	45	M	ALTERED SENSORIUM	Y	N	N	N	N	N		ALCOHOL	N	N	2	2	PANCREATITIS	PANCREATITIS	Y	N	HEPATOPANCREATOBILIARY	110
161	GS	26	M	DECREASED URINE OUTPUT	Y	N	N	N	N	Y	PARAQUAT	OPIOD	N	N	1	1	NEPHROTOXIN	NEPHROTOXIN	N	N		206
162	HC	80	M	DECREASED URINE OUTPUT	N	N	Y	N	N	N		SMOKER	N	N	0	0	INFECTION	UTI	N	N		61
163	GC	69	M	ALTERED SENSORIUM	Y	Y	N	N	N	N		N	Y	Y	3	3	INFECTION	PNEUMONIA	Y	Y	RESPIRATORY	112

S.NO	Name	UREa day 7	Serum creatinine	CREAT DAY 7	AKI STAGE	Dialysis session	SBP_DAY 0	DBP_DAY 0	SBP_DAY 7	DBP_DAY 7	Heart rate DAY 0	Heart rate DAY 7	Respiratory rate DAY 0	Respiratory rate DAY 7	Saturation oxygen DAY 0	Saturation oxygen DAY 7	TLC DAY 0	TLC DAY 7	Platelet count DAY 0	Platelet count DAY 7	Sodium DAY 0	Sodium DAY 7	Potassium DAY 0	Potassium DAY 7
126	HK	53	1.6	0.9	1	N	141	78	132	75	74	70	14	16	94	93	9	8	259	257	118	122	2.7	3.2
127	CSG	67	3	1.3	2	N	94	68	100	75	72	80	16	14	90	97	22	8	161	142	138	134	4.8	4.1
128	BS	315	6.3	5.9	3	Y	70		110	80	130	104	22	22	93	94	40	27	100	68	139	138	6.63	3.37
129	SS	151	1.7	5	1	Y	132	82	110	86	88	92	16	14	92	88	8	18	292	240	141	134	4.49	3.8
130	KL	40	2.7	1.18	2	N	126	68	130	78	100	88	20	16	88	86	21	9	91	88	138	135	4.1	3.9
131	BB	66	2.6	1.6	1	N	70	50	118	74	116	102	24	22	87	94	10	5	387	394	134	139	5.4	5.3
132	MK	22	1.8	1.31	1	N	88	62	130	70	92	90	14	16	97	98	5.8	5.2	360	349	139	138	3.9	3.8
133	MD	155	2.61	1.15	2	N	130	80	114	78	98	92	20	18	92	90	14	11	140	230	140	131	4.7	5.5
134	BS	68	1.9	1.08	1	N	110	78	122	88	102	96	18	16	95	94	18	10	326	244	138	139	2.8	3.02
135	SR	210	7.55	5.7	3	Y	122	88	114	74	99	78	22	14	84	90	10	9	139	138	139	136	4.7	4.2
136	TS	51	2	1.53	1	N	114	74	128	86	84	86	22	14	84	94	12	11	282	322	137	133	5.15	4.5
137	KK	88	1.7	3.4	1	N	100	70	104	68	89	87	14	16	94	95	29	27	738	742	114	130	6	5.4
138	SR	116	2	2.8	1	N	110	68	114	74	87	85	14	15	97	96	10	12	297	198	131	134	5.8	6.2
139	PS	72	2.7	2.6	2	N	98	68	100	75	90	88	22	14	94	93	5	5.2	110	100	130	131	3.6	3.2
140	MB	33	5	1.4	3	N	112	74	122	84	75	98	14	14	92	95	15	11	151	100	144	137	4.5	3.5
141	PK	90	2.3	1.1	2	N	122	88	127	74	78	88	16	14	97	94	11	10	422	420	144	140	3.3	4.8
142	KK	52	2	1.4	1	N	118	78	114	86	92	90	14	16	92	96	21	14	614	324	131	133	3.6	3.4
143	DS	76	2.8	1.1	2	N	94	54	88	65	72	74	18	14	90	95	16	10	80	92	136	140	5.2	4.8
144	JK	100	2.3	1.4	2	N	135	84	144	98	100	87	16	14	96	94	15	8	388	248	126	133	4.7	4.4
145	BB	123	2.14	1.7	1	N	100	70	122	78	98	85	22	18	88	90	16	11	150	181	133	141	4.3	4.4
146	SK	57	3.46	2.8	3	N	128	72	130	84	86	84	14	12	92	94	11	15	140	150	137	139	4	3.8
147	CK	28	1.9	0.8	1	N	114	78	122	84	88	90	14	16	94	95	3	8	90	110	142	141	4.3	4.1
148	JK	72	2.7	1.8	2	N	80	60	122	98	99	68	16	14	95	94	13	6	40	98	130	137	3.8	4.1
149	PK	50	1.9	1.7	1	N	112	72	124	78	102	94	20	18	94	93	6	13	158	210	141	139	4.2	4
150	HK	38	2.1	0.8	1	N	158	74	132	74	92	76	14	16	95	93	8	6	140	138	139	135	4.2	4
151	J	65	2.3	2.4	2	Y	150	80	130	70	113	122	26	24	78	74	9	8	176	154	144	138	3.8	4.2
152	H	20	1.6	0.9	1	N	130	80	122	70	88	76	18	18	96	95	20	12	343	322	137	133	3.9	3.3
153	RK	168	2.3	5.9	3	Y	100	70	110	88	133	111	26	20	85	89	14	10	120	98	126	128	5.5	4.8
154	d	126	10	7	3	Y	110	70	130	70	132	110	24	16	86	95	7	6	243	220	126	133	6.1	4.8
155	R	97	1.9	4.6	1	Y	130	90	128	76	99	76	28	24	84	77	19	18	199	178	141	140	3.1	3.2
156	V	52	4.3	1.9	3	N	140	80	128	78	122	113	21	17	89	92	8.8	10	120	118	139	142	3.6	3.7
157	R	48	2.5	0.6	2	N	110	88	130	88	132	122	26	24	90	87	30	22	386	344	149	139	5	4.4
158	B	106	4.7	3.2	3	N	88	68	128	76	98	100	22	20	92	90	14	11	222	268	136	136	5.6	4.5
159	P	74	2.3	1.4	2	N	100	70	110	78	122	100	22	16	92	88	20	18	36	44	137	133	3.9	3.8
160	BS	51	2.6	1.6	2	N	140	88	128	78	98	100	18	16	86	88	13	10	149	123	152	135	3.6	4
161	GS	227	8.3	6.5	3	Y	160	80	144	78	90	87	16	18	88	86	16	11	289	222	135	148	3.6	4.1
162	HC	45	1.6	1.9	1	N	144	88	136	78	88	94	18	16	98	96	19	14	161	110	138	151	5	5.7
163	GC	52	5.6	3.12	3	Y	88	68	100	70	77	98	26	20	79	88	24	18	197	226	134	137	4.8	4.3

S.NO	Name	AST DAY 0	AST DAY 7	ALT DAY 0	ALT DAY 7	ALP DAY 0	ALP DAY 7	Bilirubin DAY 0	Bilirubin DAY 7	Total protein DAY 0	Total protein DAY 7	Albumin DAY 0	Albumin DAY 7	Calcium DAY 0	Parathyroid (if done)	Urine R/E	UPCr(IF DONE)	Urine culture	Blood culture	Any other culture	NEUROLOGICAL FEATURES yes/no	GCS DAY 0	GCS DAY 7	Scenario 1- pt is sedated	RAAS score DAY 0
126	HK	180	112	73	48	98	92	0.5	0.4	5.7	5.5	3.3	3.2	10	9	PUS CELLS		ENTEROBACTER	N		Y	11	14	N	
127	CSG	18	16	17	15	100	112	0.18	1	5.2	6	2.9	2.8	9.4		GLUCOSURIA. PROTEINURIA					N	15	15	N	
128	BS	268	107	169	137	106	227	2.9	2.8	5.5	4.2	2.6	1.8	6.5		PUS CELLS		N	CANDIDA GILLIER		Y	6	8	Y	-1
129	SS	38	40	76	78	100	112	0.56	0.5	7.8	7.4	5.3	5.1	8.5		NORMAL					N	15	15	N	
130	KL	54	50	40	73	72	74	1.07	1	6.4	6	3.14	3	8.8		PUS CELLS	0.3	KLEBSIELLA	N		N	15	15	N	
131	BB	24	22	14	16	155	154	0.2	0.3	5.9	5.8	2.5	2.6	8.6		PUS CELLS, PROTEINURIA RBC			STAPH HEMOLYTICUS		Y	9	14	N	
132	MK	26	20	16	20	113	114	0.2	0.4	7.2	7.1	3.15	3.11	9.8		PUS CELLS		N			N	15	15	N	
133	MD	16	14	18	12	180	176	0.5	0.4	5.9	5.8	3.04	2.7	8.1		PUS CELLS RBC AND PROTEINURIA		N			N	15	15	N	
134	BS	62	68	49	54	113	118	0.8	0.9	8.3	8	4.2	3.8	8.5		PUS CELLS		N			N	15	15	N	
135	SR	13	11	12	14	126	133	1.8	2	6.9	7.01	2.8	2.7	9		PUS CELLS BACILLI	1.2	E COLI	N		N	15	15	N	
136	TS	48	66	28	31	116	106	0.42	0.3	7.4	7.1	2.9	2.4	8.8		PUS CELLS PROTEINURIA		E COLI	N	WOUND-PROTEUS KLEBSIELLA	N	15	15	N	
137	KK	28	15	13	8	497	298	1.5	0.9	5.6	5.4	2.7	2.6	7.9		NORMAL					N	15	15	N	
138	SR	22	224	68	362	52	202	1.1	1.2	5.8	5.9	2.08	2.17	8.8		PROTEINURIA					N	15	15	N	
139	PS	37	36	27	48	59	62	1.2	1.1	8	7	4.6	4.1	9.1		NORMAL					N	15	15	N	
140	MB	186	112	54	52	376	254	0.3	1	7.2	7.4	3	3.2	8		PROTEINURIA					N	15	15	N	
141	PK	24	26	12	14	81	86	0.6	0.6	7	7	2.9	2.8	14		RBC		N	N		Y	9	14	N	
142	KK	31	42	24	22	168	145	0.3	0.2	6.7	6.4	2.5	2.2	8.4		RBC		N	N	SPUTUM-KLEBSIELLA	N	15	15	N	
143	DS	172	88	89	45	228	208	3.8	2.8	6.1	5.9	3.15	3.2	9		PROTEINURIA					N	15	15	N	
144	JK	20	18	22	16	88	84	0.3	0.2	6.4	6.3	3.1	3.2	8.4		RBC					N	15	15	N	
145	BB	76	74	33	32	68	46	0.5	0.4	8	7	2.18	2	8.4		PROTEINURIA					N	15	15	N	
146	SK	35	32	48	46	566	487	0.6	0.4	5.4	4.9	3	2.9	9		PROTEINURIA					N	15	15	N	
147	CK	80	54	46	32	206	122	1.4	0.6	7.5	7	4.8	3.2	10		NORMAL					N	15	15	N	
148	JK	249	212	93	92	46	54	0.7	1	4.6	4.9	2.9	2.6	9.8		PUS CELLS		N	N		N	15	15	N	
149	PK	32	23	28	24	112	122	1.2	1.4	7	6.8	2.6	2.4	15	9/VITD -171	PUS CELLS		E COLI	N		Y	12	14	N	
150	HK	22	16	18	14	41	38	1.02	1	7.3	7.1	4.2	4.1	8.4		NORMAL					N	15	15	N	
151	J	52	88	66	98	113	143	0.15	1	7.7	6.8	5.07	3	9		PROTEINURIA					N	15	15	N	
152	H	22	20	28	31	144	122	0.9	0.6	6.1	6.6	3.7	3.3	8.8		NORMAL					N	15	15	N	
153	RK	45	42	28	30	132	112	1.1	0.9	6	6.4	2.8	2.6	13		NORMAL			N		Y	11	14	N	
154	d	98	87	199	124	126	120	0.8	0.9	5.8	2.1	6	3.2	8.5		PUS CELLS		N	N		N	15	15	N	
155	R	32	43	20	24	85	88	0.7	1.6	6.7	6.8	4.3	4.1	10		PROTEINURIA					N	15	15	N	
156	V	67	78	98	122	113	88	0.9	1.2	6.9	6	2.8	3	8.1		PROTEINURIA	0.1				N	15	15	N	
157	R	36	34	30	28	236	222	0.28	0.3	5.6	5.5	3.3	2.8	8		PUS CELLS		N	N		Y	12	15	N	
158	B	69	58	29	24	109	110	0.7	0.6	6.3	5.8	3.3	2.8	8		PUS CELLS		N	N		N	15	15	N	
159	P	29	32	27	25	426	388	3.2	2.6	5.9	6	2.5	2.6	9		PROTEINURIA					N	15	15	N	
160	BS	50	67	19	24	64	87	0.8	0.7	6	6.1	2.7	2.8	8.2		BACILLI		N	N		Y	12	14	N	
161	GS	165	166	345	409	156	165	7.9	8.1	6.3	5.9	3.6	3.3	11		RBC					N	15	15	N	
162	HC	14495	12000	3885	2888	617	566	1.9	2	4	5	5	2.05	9		PUS CELLS		E COLI	N		N	15	15	N	
163	GC	88	84	98	96	112	145	0.6	0.8	6.1	5.8	2.3	2.5	8.6		PROTEINURIA		N	N	ET-ACINETOBACTER FUNGITELL	Y	11	12	N	

S.NO	Name	RAAS score DAY 7	PATIENT IS DELIRIOUS	CAM ICU DAY 0	CAM ICU DAY 7	MMSE DAY 0	MMSE DAY 7	SERIAL 7_DAY 0	SERIAL 7 _DAY 7	MYCOLONUS DAY 0	MYCOLONUS DAY 7	DELUSION/HALLUCINATION DAY 0	DELUSION/HALLUCINATION DAY 7	Asterixis DAY 0	Asterixis DAY 7	Seizure DAY 0	Seizure DAY 7	Sleep inversion DAY 0	Sleep inversion DAY 7	aphasia DAY 0	aphasia DAY 7	cranial nerve involved	MUSCULAR DISORDERSDAY 0	MUSCULAR DISORDERS DAY 7	Tremor/involuntary movements DAY 0
126	HK		N	N	N	4	8	0	1	N	N	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N
127	CSG		N	N	N	27	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
128	BS	1	N	N	N	0	0	0	1	Y	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N
129	SS		N	N	N	26	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
130	KL		N	N	N	29	29	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
131	BB		Y	Y	N	0	14	0	1	Y	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N
132	MK		N	N	N	28	28	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
133	MD		N	N	N	27	29	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
134	BS		N	N	N	26	26	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
135	SR		N	N	N	29	29	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
136	TS		N	N	N	27	28	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
137	KK		N	N	N	28	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
138	SR		N	N	N	29	28	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
139	PS		N	N	N	28	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
140	MB		N	N	N	27	28	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
141	PK		Y	Y	Y	0	10	0	2	N	N	Y	N	N	N	N	N	Y	Y	N	N	N	N	N	N
142	KK		N	N	N	27	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
143	DS		N	N	N	27	29	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
144	JK		N	N	N	28	27	3	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
145	BB		N	N	N	27	28	3	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
146	SK		N	N	N	27	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
147	CK		N	N	N	28	27	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
148	JK		N	N	N	27	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
149	PK		Y	Y	N	0	12	0	2	Y	N	Y	N	Y	N	N	N	N	N	N	N	N	N	N	Y
150	HK		N	N	N	27	27	3	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
151	J		N	N	N	28	30	5	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
152	H		N	N	N	29	29	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
153	RK		Y	Y	N	0	0	0	1	N	N	N	N	Y	N	N	N	Y	N	N	N	N	N	N	Y
154	d		N	N	N	27	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
155	R		N	N	N	28	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
156	V		N	N	N	28	27	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
157	R		Y	Y	N	0	14	0	2	N	N	Y	N	Y	Y	Y	N	Y	Y	N	N	N	N	N	Y
158	B		N	N	N	27	28	4	3	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
159	P		N	N	N	28	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
160	BS		Y	Y	N	0	0	0	0	N	N	Y	N	Y	Y	N	Y	Y	Y	N	N	N	N	N	Y
161	GS		N	N	N	27	28	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
162	HC		N	N	N	29	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
163	GC		Y	Y	Y	0	0	0	0	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N

S.NO	Name	Tremor/involuntary movements DAY 7	Wasting DAY 0	Wasting DAY 7	Fasciculation DAY 0	Fasciculation DAY 7	SENSORY SYSTEM	ABNORMAL DEEP TENDON reflexes	LOSS OF URINARY CONTROL DAY 0	LOSS OF URINARY CONTROL DAY 7	Night diarrhea DAY 0	Night diarrhea DAY 7	Neck rigidity DAY 0	Neck rigidity DAY 7	ADDITIONAL INVESTIGATIONS AS AND WHEN INDICATED_CT HEAD	MRI	EEG	CSF ANALYSIS-as and when required	USG abdomen
126	HK	N	N	N	N	N	N	N	N	N	N	N	N	N	NORMAL	FRONTOTEMPORALDEMENTIA	X	X	CYSTITIS
127	CSG	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
128	BS	N	N	N	N	N	N	N	N	N	N	N	N	N	NORMAL	CYTOTOXIC EDEMA BILATERAL SEMIOVALE	X	X	FATTY LIVER ASCITES
129	SS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ACUTE RENAL DISEASE
130	KL	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CLD
131	BB	N	N	N	N	N	X	X	N	N	N	N	N	N	NORMAL	X	X	X	GB EDEMA
132	MK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	LEFT RENAL INFARCT
133	MD	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
134	BS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
135	SR	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HYDROURETRONEPHROSIS
136	TS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CYSTITIS
137	KK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HEPATOSPLENOMEGALY
138	SR	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
139	PS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB SLUDGE
140	MB	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	SPLENOMEGALY
141	PK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
142	KK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PERINEPHRIC STRANDING
143	DS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ASCITES
144	JK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
145	BB	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ASCITES
146	SK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HEPATOMEGALY
147	CK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
148	JK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	SPLENOMEGALY
149	PK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CYSTITIS
150	HK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
151	J	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	RENAL PARENCHYMAL CHANGES
152	H	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
153	RK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	SYMMETRICAL HYPERINTENSITY IN BILATERAL GLOBUS PALLIDUS AND DORSAL PORTION OF BILATERAL PUTAMINA.	X	X	RENAL CHANGES
154	d	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PERINEPHRIC STARNDING
155	R	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CHOLELITHIAISIS
156	V	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
157	R	Y	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PANCREATITIS
158	B	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HYDRONEPHROSIS
159	P	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PERIPANCREATIC STRANDING
160	BS	Y	N	N	N	N	X	N	N	N	N	N	N	N	NORMAL	X	X	X	PANCREAS BULKY
161	GS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ACUTE RENAL DISEASE
162	HC	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CYSTITIS
163	GC	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CHOLELITHIASIS

S.NO	Name	Age	Sex	Presenting complaints	ICU STAY	hypertension	diabetes mellitus 2	CLD	CAD	Any nephrotoxic drugs prior to admission	Type of nephrotoxic drugs	H/O Alcohol/smoking/substance abuse	Mechanical ventilatory support	Ionotropic support	qSOFA _Day 0	qSOFA _Day 7	CAUSE	AKI etiology	sepsis present	septic shock present	source of sepsis	UREA DAY 0
164	D	65	M	DECREASED URINE OUTPUT	Y	N	N	N	N	N		ALCOHOL	N	Y	3	1	INFECTION	PYELONEPHRITIS	Y	Y	UROSEPSIS	52
165	M	20	M	DECREASED URINE OUTPUT	Y	N	N	N	N	Y	PARAQUAT	N	N	N	3	3	NEPHROTOXIN	NEPHROTOXIN	N	N		240
166	AK	65	F	DYSPNEA	N	Y	N	N	N	N		N	N	N	0	0	INFECTION	PNEUMONIA	N	N		59
167	KD	65	F	PAIN ABDOMEN	N	Y	Y	N	N	N		N	N	N	1	1	INFECTION	ACUTE CHOLANGITIS	N	N		62
168	P	79	M	PAIN ABDOMEN	N	N	N	Y	N	N		SMOKING	N	N	0	0	INFECTION	UTI	N	N		36
169	SK	85	F	PEDAL EDEMA	N	N	N	N	N	N		N	N	N	0	0	MISC	MALIGNANCY	N	N		87
170	PS	27	M	YELLOWING SCLERA	N	N	N	N	N	N		ALCOHOL	N	N	0	0	INFECTION	VIRAL HEPATITIS	N	N		171
171	S	41	F	SNAKE BITE	Y	N	N	N	N	N		N	N	N	0	0	MISC	SNAKE BITE	N	N		194
172	AS	62	M	CONSTIPATION	N	N	Y	N	N	N		N	N	N	0	0	SURGICAL	INTESTINAL OBTSRUCTION	N	N		62
173	S	49	F	DECREASED URINE OUTPUT	N	N	Y	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		68
174	N	55	F	FEVER AND ALTERED SENSORIUM	Y	N	N	N	N	N		N	N	Y	2	2	TROPICAL FEVER	SCRUB TYPHUS	Y	Y	RESPIRATORY	55
175	ID	58	M	FEVER	N	N	N	N	N	N		N	N	N	0	0	INFECTION	PYELONEPHRITIS	N	N		193
176	M	18	F	FEVER	N	N	N	N	N	N		N	N	N	0	0	TROPICAL FEVER	LEPTOSPIRA, SCRUB TYPHUS	N	N		99
177	B	45	M	LOOSE STOOLS	N	N	N	N	N	N		ALCOHOL	N	N	2	2	MISC	HIV	N	N		99
178	J	31	F	LOWER BACK PAIN	Y	Y	N	N	N	N		N	N	Y	2	0	INFECTION	PYOMYOSITIS,PSOAS ABCESS	Y	N	PSOAS ABCESS	215
179	G	61	F	RIGHT LOWER LIMB SWELLING	N	N	N	N	N	N		N	N	N	2	1	INFECTION	CELLULITIS	Y	N	CELLULITIS	112
180	PS	22	M	FEVER	N	N	N	N	N	N		N	N	N	0	0	TROPICAL FEVER	SCRUB TYPHUS	N	N		100
181	B	68	F	PAIN ABDOMEN	N	N	N	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		58
182	KK	54	F	LOOSE STOOLS	N	N	N	N	N	N		N	N	N	0	0	MISC	MULTIPLE MYELOMA	N	N		129
183	DS	73	M	LOOSE STOOLS	N	Y	Y	N	Y	N		ALCOHOL	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		104
184	BM	72	M	HEAD ACHE	N	N	N	N	N	N		N	N	N	0	0	INFECTION	FUNGAL PNEUMONIA	N	N		198
185	JK	63	F	DYSPNEA	N	Y	N	N	N	N		N	N	N	0	0	INFECTION	PNEUMONIA	N	N		101
186	VS	55	M	DECREASED URINE OUTPUT	Y	N	N	N	N	Y	CELPHOS	ALCOHOL	N	N	0		NEPHROTOXIN	NEPHROTOXIN	N	N		78
187	HK	70	F	PAIN ABDOMEN	Y	N	Y	N	Y	N		N	N	Y	1	1	PANCREATITIS	PANCREATITIS	N	N		72
188	RS	70	M	ABNORMAL BODY MOVEMENTS	Y	N	N	N	N	Y	NSAIDS	N	N	N	2	1	INFECTION	VIRAL HEPATITIS	N	N		84
189	R	55	F	LOWER LIMB WEAKNESS	Y	N	N	N	N	N		N	N	N	1	1	INFECTION	TUBERCULOSIS	N	N		84
190	SS	40	M	PAIN ABDOMEN	N	N	N	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		110
191	N	25	M	BURNING MICTURITION	N	N	N	N	N	N		N	N	N	0	0	INFECTION	UTI	N	N		131
192	JS	40	M	TREMORS	N	N	N	N	N	N		ALCOHOL	N	N	1	1	HYPOVOLUMEA	HYPOVOLUMEA	N	N		46
193	SS	80	M	HEMATURIA	N	Y	Y	N	N	N		ALCOHOL	N	N	0	1	OBSTRUCTIVE	HYDROURETRONEPHROSIS	N	N		541
194	L	24	M	DECREASED URINE OUTPUT	Y	N	N	N	N	Y	VANCOMYCIN	N	N	N	1	0	INFECTION	INFECTIVE ENDOCARDITIS	N	N		92
195	SR	89	F	LOOSE STOOLS	N	N	Y	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		68
196	NJ	56	M	VOMITING	N	N	N	N	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		64
197	PK	42	F	PAIN ABDOMEN, MISSED LMP	Y	N	N	N	N	N		N	N	Y	2	1	SURGICAL	ECTOPIC PREGNANCY	Y	Y	ECTOPIC PREGNANCY	53
198	NS	70	M	BURNING MICTURITION	Y	Y	Y	Y	Y	N		N	N	Y	2	1	INFECTION	PYELONEPHRITIS	Y	Y	UROSEPSIS	198
199	SK	55	F	LOOSE STOOLS	N	N	N	Y	N	N		N	N	N	0	0	INFECTION	ACUTE GASTROENTERITIS	N	N		220
200	VD	68	F	LOOSE STOOLS	N	N	N	N	Y	N		N	N	Y	2	1	INFECTION	ACUTE GASTROENTERITIS	Y	N	GASTROENTEROLOGY	48

S.NO	Name	UREa day 7	Serum creatinine	CREAT DAY 7	AKI STAGE	Dialysis session	SBP_DAY 0	DBP_DAY 0	SBP_DAY 7	DBP_DAY 7	Heart rate DAY 0	Heart rate DAY 7	Respiratory rate DAY 0	Respiratory rate DAY 7	Saturation oxygen DAY 0	Saturation oxygen DAY 7	TLC DAY 0	TLC DAY 7	Platelet count DAY 0	Platelet count DAY 7	Sodium DAY 0	Sodium DAY 7	Potassium DAY 0	Potassium DAY 7
164	D	62	3.1	2.07	2	Y	128	78	98	68	99	86	20	16	90	91	20	12	177	32	148	129	4.2	4.3
165	M	118	7.2	1.6	3	Y	130	78	122	68	92	98	22	16	92	90	17	16	118	114	142	144	5	5.2
166	AK	28	1.6	0.6	1	N	100	60	110	72	99	91	22	14	88	92	6.4	5	164	188	126	135	6.5	3.7
167	KD	77	2.24	2.4	2	N	112	78	130	88	90	88	18	16	94	92	8	6	192	141	140	139	4.9	4.08
168	P	34	1.7	1.2	1	N	122	78	132	88	98	92	19	16	94	95	3.2	3.6	48	52	134	141	4.4	4.2
169	SK	66	1.8	1.37	1	N	110	78	132	98	78	82	18	20	94	93	5	6	131	115	132	131	3.6	3.5
170	PS	172	8.3	5.6	3	N	132	78	100	88	77	65	16	14	92	90	9	10	333	302	133	132	4.4	4.5
171	S	200	8.74	8.22	3	Y	110	78	112	99	92	90	16	14	94	93	17	13	120	158	124	126	4.25	4.3
172	AS	112	4.7	3.3	3	N	100	79	122	87	88	90	14	16	98	97	11	14	144	158	134	143	4	5
173	S	45	1.8	0.6	1	N	129	87	110	78	69	76	18	14	94	93	15	16	155	164	143	143	3.1	4.9
174	N	242	1.9	5.6	1	Y	112	78	133	88	78	90	18	14	90	88	10	14	50	30	141	140	4.2	4.1
175	ID	114	7	3	3	N	145	78	144	68	88	78	16	14	95	94	17	9	300	326	136	137	4.6	4.1
176	M	100	1.61	1.01	1	N	110	78	133	88	95	93	14	16	98	95	10	9	50	40	141	140	4	4.3
177	B	96	3.6	7.2	3	N	110	78	100	88	80	78	14	16	92	90	21	16	264	266	141	144	4.1	4.5
178	J	52	4.4	1.6	3	N	98	65	100	80	76	80	22	17	94	95	20	15	78	45	133	136	4	4.6
179	G	40	2.6	0.6	2	N	96	58	98	68	84	82	24	16	88	92	22	18	98	78	147	144	3.5	4
180	PS	98	1.7	0.8	1	N	130	78	122	87	87	86	14	15	99	97	8	7	110	150	148	144	3.6	4.1
181	B	60	1.9	1.2	1	N	98	72	100	60	88	98	16	15	98	88	5	9	176	180	140	138	4	4.2
182	KK	160	7	8	3	Y	99	67	89	66	90	92	14	15	95	96	7	7	72	89	134	133	5	4.5
183	DS	46	1.8	1.1	1	N	110	82	98	72	95	94	16	14	98	97	6	6	64	110	138	142	4.3	4
184	BM	101	5	4.2	3	Y	111	72	100	85	96	78	16	14	96	94	7	9	88	98	136	135	3.8	3.5
185	JK	88	1.9	1.4	1	N	96	70	100	67	74	78	14	14	95	92	12	8	362	284	142	140	4.7	4.1
186	VS		1.6	1.1	1	N	140	90	110	88	66		18		90		11		142		138		4.5	
187	HK	57	1.5	0.9	1	N	92	66	98	76	66	68	14	17	96	94	12	19	140	237	146	139	4.5	4.1
188	RS	172	3.7	5.6	3	N	110	78	104	74	122	100	12	16	98	95	13	23	80	36	133	131	4.1	3.6
189	R	90	2.2	1.4	2	N	156	98	142	74	67	72	14	16	94	93	30	9	90	40	133	139	5.5	5.1
190	SS	120	2.1	2.4	1	N	100	74	132	98	98	92	16	18	94	95	16	14	152	144	135	136	4.8	5
191	N	133	4.2	4.05	3	N	102	78	116	78	102	86	14	16	92	90	14	12	38	34	132	142	5	4.8
192	JS	35	1.6	1	1	N	144	78	122	68	98	94	14	12	95	93	8	11	192	156	135	133	3.8	4.1
193	SS	288	7.6	4.5	3	Y	177	100	145	98	75	68	22	16	88	93	6.8	6.5	268	234	142	144	6.1	5.2
194	L	54	1.8	1.4	1	N	100	78	112	88	87	94	18	14	93	95	38	42	38	25	142	136	4.5	4.7
195	SR	59	1.6	0.9	1	N	168	88	150	82	88	78	14	16	97	96	12	15	240	283	137	128	5.1	3.8
196	NJ		1.6		1	N	122	78			98		15		93		9		220		142		3.5	
197	PK	65	3	3.15	2	N	94	64	102	78	100	94	22	16	90	97	14	19	150	217	137	139	4.3	3.18
198	NS	294	6	6.9	3	Y	88	56	110	78	112	102	22	16	90	94	46	17	40	45	135	132	5.2	5
199	SK	90	7.8	2.3	3	Y	132	78	128	74	94	84	14	16	95	94	7	7	159	142	126	134	4.2	3.45
200	VD	43	1.5	1.15	1	N	64	32	88	54	100	94	16	14	92	90	12.8	13	434	352	135	144	5.6	3.9

S.NO	Name	AST DAY 0	AST DAY 7	ALT DAY 0	ALT DAY 7	ALP DAY 0	ALP DAY 7	Bilirubin DAY 0	Bilirubin DAY 7	Total protein DAY 0	Total protein DAY 7	Albumin DAY 0	Albumin DAY 7	Calcium DAY 0	Parathyroid (if done)	Urine R/E	UPCr(IF DONE)	Urine culture	Blood culture	Any other culture	NEUROLOGICAL FEATURES yes/no	GCS DAY 0	GCS DAY 7	Scenario 1- pt is sedated	RAAS score DAY 0
164	D	35	45	26	22	88	132	1.03	0.6	5.8	6.1	3.2	2.8	9.1		PUS CELLS RBC+BL175		N	N		Y	11	13	N	
165	M	151	254	310	308	448	424	4.6	4.4	6	5.8	3.3	3.1	10		PUS CELLS		N	N		N	15	15	N	
166	AK	24	18	19	16	111	98	0.1	0.2	5.7	5.5	2.8	2.9	9.2		NORMAL					N	15	15	N	
167	KD	16	14	17	16	60	57	0.2	0.9	6.1	5.8	3.3	3	9.2		NORMAL					N	15	15	N	
168	P	35	32	10	18	112	105	1	1	6	5	2.6	2.5	10		NORMAL					N	15	15	N	
169	SK	17	18	12	13	97	96	0.45	0.5	5.4	5.4	1.9	2	10.4		PUS CELLS		N	N		N	15	15	N	
170	PS	104	114	572	540	397	392	10	9	6.8	6.4	3.2	3.1	9.8		NORMAL					N	15	15	N	
171	S	45	29	42	54	54	59	1.2	1.07	6	5.8	3.1	2.8	9.9		PROTEINURIA	0.4				N	15	15	N	
172	AS	33	78	133	144	65	88	0.8	1	6	5.9	3	3	9		NORMAL					N	15	15	N	
173	S	334	325	125	128	223	125	0.9	0.8	6	5.7	3	2.9	9.9		NORMAL					N	15	15	N	
174	N	164	78	67	45	271	188	2.6	0.6	5.6	6.2	2.5	2.6	10		PUS CELLS		CANDIDA	N		Y	11	10	N	
175	ID	80	56	90	44	110	114	0.8	1	6	6	2.5	2.6	9.8		PUS CELLS	1	CANDIDA	N		N	15	15	N	
176	M	77	75	61	60	456	424	1.3	1.1	6	5.8	2.5	2.6	10		NORMAL					N	15	15	N	
177	B	64	92	88	86	100	135	2	1	6	5	2.6	3	9		RBC					N	15	15	N	
178	J	132	110	78	72	99	144	4.9	2	5	6	3.5	4	9.2		PUS CELLS		N	N		N	15	15	N	
179	G	148	128	112	188	198	200	1.2	1.4	7	6	3	3.4	9		PROTEINURIA					N	15	15	N	
180	PS	138	137	112	133	577	324	2.2	2	6.8	6	3.7	3.2	8		YEAST		N	N		N	15	15	N	
181	B	20	20	20	18	88	100	1.2	0.9	6	5.8	3	2.8	9		NORMAL					N	15	15	N	
182	KK	72	78	74	88	83	99	1.5	1.6	5.6	6	2.5	2.6	8.4		PROTEINURIA					N	15	15	N	
183	DS	88	86	82	80	42	38	0.9	1	6.2	6	2.1	2.3	9		RBC	1				N	15	15	N	
184	BM	55	45	89	67	54	52	0.2	0.1	6	6.8	2.8	2.4	8.4		PUS CELLS		N	N		N	15	15	N	
185	JK	18	16	14	12	108	100	0.4	0.52	7.6	7.2	3.9	3.4	7		PROTEINURIA					N	15	15	N	
186	VS	122		154		144		1.2		7.4		3.8		8.4		RBC					N	15	15	N	
187	HK	98	92	115	98	96	68	1	2.1	7.1	6.8	3.4	3	9.2		RBC PROTEINURIA		N	E COLI		N	15	15	N	
188	RS	14000	10555	15222	9222	654	466	14	22	4.8	2.8	5.3	3	8.5		RBC		N	N		Y	11	10	N	
189	R	70	131	18	40	174	160	5.2	3.1	5.3	5.6	2.2	2.1	8.4		NORMAL		N	N		Y	12	11	N	
190	SS	92	80	76	68	112	115	7	6	3	2.9	3	4	9		PUS CELLS		N	N		N	15	15	N	
191	N	135	637	73	80	234	291	8.9	8.3	6	5	2.2	2.3	8.1		PROTEINURIA					N	15	15	N	
192	JS	28	42	23	18	107	100	0.3	0.2	7.4	6.5	3.1	2.8	8		NORMAL					Y	11	15	N	
193	SS	23	21	13	14	62	66	5.5	5.8	6	6.4	2.7	2.5	8.4		PUS CELLS		N	N			11	13	N	
194	L	96	95	22	28	244	109	4.7	4.6	5.8	6	2.6	3	9		NORMAL		N	MRSA		N	15	15	N	
195	SR	28	26	12	14	92	68	0.2	0.2	6	6.7	3	2.9	8.1		PUS CELLS		N	N		N	15	15	N	
196	NJ	35		52		112		0.9		7		3		8.9		NORMAL					N	15	15	N	
197	PK	251	242	19	17	316	255	8.8	6	5.3	6	2.9	2.8	8.7		PUS CELLS PROTEINURIA			N		N	15	15	N	
198	NS	55	54	34	31	196	177	4.9	5.6	5.3	6.1	1.9	2.2	9		PUS CELLS		KLEBSIELLA	N		N	15	15	N	
199	SK	18	16	11	14	66	68	0.9	1	7.8	6.9	2.8	3	7		NORMAL					N	15	15	N	
200	VD	17	16	7	8	82	80	1.4	1.1	7	6	2.5	2.1	6.5		NORMAL					N	15	15	N	

S.NO	Name	RAAS score DAY 7	PATIENT IS DELIRIOUS	CAM ICU DAY 0	CAM ICU DAY 7	MMSE DAY 0	MMSE DAY 7	SERIAL 7_DAY 0	SERIAL 7 _DAY 7	MYCOLONUS DAY 0	MYCOLONUS DAY 7	DELUSION/HALLUCINATION DAY 0	DELUSION/HALLUCINATION DAY 7	Asterixis DAY 0	Asterixis DAY 7	Seizure DAY 0	Seizure DAY 7	Sleep inversion DAY 0	Sleep inversion DAY 7	aphasia DAY 0	aphasia DAY 7	cranial nerve involved	MUSCULAR DISORDERSDAY 0	MUSCULAR DISORDERS DAY 7	Tremor/involuntary movements DAY 0
164	D		Y	Y	Y	0	0	0	0	Y	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	N
165	M		N	N	N	26	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
166	AK		N	N	N	26	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
167	KD		N	N	N	28	29	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
168	P		N	N	N	28	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
169	SK		N	N	N	28	29	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
170	PS		N	N	N	29	29	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
171	S		N	N	N	29	28	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
172	AS		N	N	N	27	28	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
173	S		N	N	N	29	27	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
174	N		Y	Y	Y	0	0	0	0	N	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	N
175	ID		N	N	N	29	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
176	M		N	N	N	30	29	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
177	B		N	N	N	28	29	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
178	J		N	N	N	29	28	3	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
179	G		N	N	N	28	30	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
180	PS		N	N	N	30	30	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
181	B		N	N	N	29	28	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
182	KK		N	N	N	29	28	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
183	DS		N	N	N	28	27	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
184	BM		N	N	N	26	26	5	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
185	JK		N	N	N	29	27	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
186	VS		N	N		27		4		N		N		N		N		N		N		N	N	N	N
187	HK		N	N	N	28	29	4	4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
188	RS		Y	Y	Y	0	0	0	0	Y	N	Y	N	Y	N	Y	N	Y	N	N	N	N	N	N	Y
189	R		Y	Y	N	0	0	0	0	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	N
190	SS		N	N	N	27	28	3	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
191	N		N	N	N	27	28	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
192	JS		N	N	N	0	20	0	4	N	N	Y	N	Y	N	Y	N	Y	N	N	N	N	N	N	Y
193	SS		Y	Y	Y	0	0	0	0	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N
194	L		N	N	N	28	29	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
195	SR		N	N	N	27	29	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
196	NJ		N	N		29		4		N		N		N		N		N		N		N	N		N
197	PK		N	N	N	28	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
198	NS		N	N	N	26	27	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
199	SK		N	N	N	27	26	4	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
200	VD		N	N	N	28	29	3	5	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N

S.NO	Name	Tremor/involuntary movements DAY 7	Wasting DAY 0	Wasting DAY 7	Fasciculation DAY 0	Fasciculation DAY 7	SENSORY SYSTEM	ABNORMAL DEEP TENDON reflexes	LOSS OF URINARY CONTROL DAY 0	LOSS OF URINARY CONTROL DAY 7	Night diarrhea DAY 0	Night diarrhea DAY 7	Neck rigidity DAY 0	Neck rigidity DAY 7	ADDITIONAL INVESTIGATIONS AS AND WHEN INDICATED_CT HEAD	MRI	EEG	CSF ANALYSIS-as and when required	USG abdomen
164	D	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	RENAL CALCULI
165	M	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ACUTE RENAL DISEASE
166	AK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
167	KD	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	OBSTRUCTIVE BILIOPATHY
168	P	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CLD/PORTAL HTN
169	SK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ASCITES
170	PS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HEPATITIS
171	S	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ASCITES
172	AS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HEPATOSPLENOMEGALY
173	S	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ASCITES
174	N	N	N	N	N	N	X	N	N	N	N	N	N	N	N	NORMAL	X	X	FATTY LIVER RENAL CHANGES
175	ID	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HYDRONEPHROSIS
176	M	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB POLYP
177	B	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
178	J	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HEPATOSPLENOMEGALY
179	G	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
180	PS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
181	B	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HEPATOMEGALY
182	KK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	NORMAL
183	DS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CHOLELITHIASIS
184	BM	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CYSTITIS
185	JK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CHOLELITHIASIS
186	VS		N		N		N	N	N		N		N		X	X	X	X	NORMAL
187	HK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ASCITES
188	RS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HEPATITIS, GB EDEMA
189	R	N	N	N	N	N	N	N	N	N	N	N	N	N	HYDROCEPHALUS	X	X	X	CLD
190	SS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HEPATOSPLENOMEGALY
191	N	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
192	JS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	FATTY LIVER
193	SS	N	N	N	N	N	N	N	N	N	N	N	N	N	NORMAL	X	X	X	HYDRONEPHOSIS
194	L	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	ASCITES
195	SR	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	CYSTITIS
196	NJ		N		N		N	N	N		N		N		X	X	X	X	NORMAL
197	PK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	RUPTURED ECTOPIC PREGNANCY
198	NS	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	PYELONEPHRITIS
199	SK	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	GB EDEMA
200	VD	N	N	N	N	N	N	N	N	N	N	N	N	N	X	X	X	X	HEPATOSPLENOMEGALY

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Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.10.2021 has approved the plan of thesis of **Arshdeep Kaur Gill**, a student of your college for **MD General Medicine** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis

Spectrum of neurological manifestations in acute kidney injury: a prospective observational study in tertiary care centre.

Supervisor
Dr. Sandeep Chhabra
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The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

41. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*
42. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

SPECTRUM OF NEUROLOGICAL MANIFESTATIONS IN ACUTE KIDNEY INJURY: A PROSPECTIVE OBSERVATIONAL STUDY IN TERTIARY CARE CENTRE

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

MD

MEDICINE

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

MARCH 2021 **DR ARSHDEEP KAUR GILL**

DEPARTMENT OF MEDICINE
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	Spectrum of neurological manifestations in Acute kidney injury: A prospective observational study in tertiary care centre
For the degree of	MEDICINE
Name of the candidate	Dr Arshdeep Kaur Gill
Supervisor	Dr Sandeep Chhabra, Associate Professor, Department of Medicine, DMC&H Ludhiana
Co-supervisor	Dr Monika Singla, Associate Professor, Department of Neurology, DMC&H Ludhiana Dr Suman Sethi, Assistant Professor, Department of Nephrology, DMC&H, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Acute kidney injury is one of the most important causes of morbidity and mortality with a myriad of clinical spectrum including cardiovascular, hematological, neurological and respiratory complications. These changes include headache, visual abnormalities, myoclonus, chorea, seizures, cerebrovascular accident, peripheral neuropathies and coma.

Aim of Study: To assess the spectrum of neurological features in Acute kidney injury and correlate it to stages of Acute kidney injury and underlying etiology.

Materials & methods: A prospective observational study will be conducted on 200 patients who have Acute kidney injury. A detailed clinical examination including GCS will be documented along with blood counts, chemistry, ABG supplemented by MRI CT EEG NCV AND CSF analysis as and when required

Clinical Significance: Neurological features in Acute kidney injury are more abrupt in onset and more severe. the signs and symptoms vary depending on biological characterstic of the patient , underlying cause and timing of injury. The symptoms of Acute kidney injury are readily relieved by dialysis. Early recognition of neurological changes can help improve clinical outcome.

KEYWORDS: Acute kidney injury, neurological, creatinine, urine output, sepsis, delirium, seizures, dialysis, stroke, GCS, uremia.

(Signature)	(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-supervisor	Co-supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"SPECTRUM OF NEUROLOGICAL MANIFESTATIONS IN ACUTE KIDNEY INJURY: A PROSPECTIVE OBSERVATIONAL STUDY IN TERTIARY CARE CENTRE"** exist in the Department of General Surgery, DMC&H, Ludhiana and will be provided to be candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate himself under our supervision and guidance.

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APPROVAL PROFORMA
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Name of candidate	Dr. Arshdeep Kaur Gill
Department	Medicine
Topic of Thesis	SPECTRUM OF NEUROLOGICAL MANIFESTATIONS IN ACUTE KIDNEY INJURY: A PROSPECTIVE OBSERVATIONAL STUDY IN TERTIARY CARE CENTRE
Likely date of appearing in PG examination	MAY 2023
Date of enrollment	26th July 2020
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Approved: Yes / No

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Approved: Yes / ~~No~~

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BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

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4	DATE OF ADMISSION OF COURSE	26 JULY 2020
5	TITLE OF THE TOPIC	SPECTRUM OF NEUROLOGICAL MANIFESTATIONS IN ACUTE KIDNEY INJURY:A PROSPECTIVE OBSERVATIONAL STUDY IN TERTIARY CARE CENTRE

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Acute kidney injury is one of the most common complication associated with remote organ dysfunction in critically ill patients. Initially wide array of definitions were used to define acute renal failure but a consensus was decided to follow KDIGO criteria which uses the surrogates of kidney function, serum creatinine level and urine output, to define the presence and severity of kidney injury[1] The incidence of Acute kidney injury is 21.6 % in adults with mortality of 23.9 % in adults[2] It has been found that Acute kidney injury is more likely to occur in children and healthy adults in developing countries than in developed countries because of a host of socioeconomic and environmental influences [3] Acute kidney injury can be hospital acquired or community acquired. The causes of Acute kidney injury show wide variation in developed and developing countries. Factors contributing to Acute kidney injury such as infectious diseases (that is, tropical febrile illnesses such as diarrhea, leptospirosis, malaria and dengue fever), envenoming (as a result of snake, spider or insect bites and/or stings), and obstetrical complications are described far more commonly in developing countries than in those from developed countries [4]. The factors that contribute to Acute kidney injury-associated mortality vary with time and differ in the immediate and long-term. Factors that modify the risk of early mortality occurring in-hospital or less than 90 days after Acute kidney injury include the primary diagnosis (such as sepsis), severity of acute illness and the burden of acute nonrenal organ dysfunction[5] Mortality during Acute kidney injury is largely due to involvement of non renal vital organ Complex mechanisms of crosstalk between injured kidneys and remote organs such as the nervous system, lungs, liver, heart, gut, brain, and hematologic system have been identified. Central nervous system changes including decreased mental status, obtundation and seizures, are one of the classic indications to begin dialysis during Acute kidney injury. Dialysis improves but does not fully correct central nervous system manifestations or other distant organ effects of renal failure, either acutely or chronically.[6] The neurological

manifestations in Chronic kidney disease have been well described in studies including stroke, intracerebral microbleeds and cognitive [7,8]. Few studies have evaluated the neurological features in Acute kidney injury even though the symptoms of encephalopathy are generally more pronounced and progress more rapidly in Acute kidney injury than with chronic kidney disease or ESRD.[9]

While patients with acute renal failure suffer from mainly Central nervous system symptoms, uremic encephalopathy has become much less common in Chronic kidney disease with use of renal replacement. Patients with Acute kidney injury also develop confusion, headache, visual abnormalities, tremors, asterixis, chorea, seizures and coma, apart from this there is an increased risk of drug induced encephalopathy. Several mechanisms have been proposed for the neurological manifestations of Acute kidney injury. Uremic toxins certainly contribute to many symptoms of encephalopathy however, cellular and soluble inflammatory mediators have also been implicated. an increase of KC, G-CSF, and glial fibrillary acidic protein (GFAP) in the cerebral cortex and hippocampus of the brain may serve to recruit neutrophils to sites of neuronal damage[10]

Identifying the neurological features in Acute kidney injury can help stratify patients for management and reduce mortality and morbidity. Chronic kidney disease has been widely studied for its neurological spectrum but Acute kidney injury is uncharted waters in this respect. Isolated studies have been done on neurological features specific to etiology but no study has been done in india to analyse the neurological aspect of Acute kidney injury as a single entity. This would be the first study to assess neurological features of Acute kidney injury irrespective of underlying cause in India and use the data to draw inference about the clinical presentation and correlation if present with disease outcome.

6.2 REVIEW OF LITERATURE

Acute renal failure results from a rapid decline in glomerular filtration rate accompanied by accumulation of nitrogenous waste products and disturbances of fluid, electrolyte, and acid-base homeostasis. Neurologic complications of renal failure can involve the Central nervous system, peripheral nervous system, or both. They can occur as a direct consequence of the uremic state, its treatment, or both.

The term uremic encephalopathy is used to describe the early appearance and dialysis responsiveness of the nonspecific neurologic symptoms of uremia. The symptoms of uremic encephalopathy may include sluggishness and easy fatigue; daytime drowsiness and insomnia with a tendency toward sleep-inversion; itching; inability to focus or sustain attention or to perform mental tasks; inability to manage ideas and abstractions; slurring of speech; anorexia, nausea, and vomiting; restlessness; imprecise memory; disorientation and confusion with bizarre behavior.

Uremic symptoms are generally more severe and progress more rapidly in patients with acute renal failure than in those with chronic renal failure. The symptoms of acute renal failure are readily relieved by dialysis procedures.

Dialysis itself is associated with neurological syndromes including the dysequilibrium syndrome, subdural haematoma, and Wernicke's encephalopathy.

Abnormalities of mental status have been noted to be sensitive indices of acute renal insufficiency, which may progress rapidly into disorientation and confusion if untreated. Fixed attitudes, torpor, and other signs of toxic psychosis are common. untreated it can progress to coma Cranial nerve signs such as nystagmus and mild facial asymmetries are usually transient. Asymmetrical variation of deep tendon reflexes is noted along with progression of hyperreflexia, with sustained clonus.

This review will focus on the neurological manifestations which have been found to occur in association with Acute kidney injury. It will also

explore the radiological and EEG surrogates of the neurological changes.

Uremic encephalopathy occurs when glomerular filtration rate declines. Lee et al found that A baseline eGFR <60 ml/min/1.73 m^2 was independently related to incident stroke[11] Paul Teschan et al observed that there are quantitative impairments of Central nervous system function that vary directly with the degree of renal failure, improve following onset of maintenance dialysis, and improve still further after successful renal transplantation.[12]

Sepsis is the most common cause of Acute kidney injury worldwide. It can be secondary to infections of skin/soft tissue/lung/endocarditis in order of percentage share. Tropical fevers form a major share of Acute kidney injury in developing countries especially india.[13]

Shukla et al found that (21.8% of patients with malaria associated Acute kidney injury presented with cerebral changes. [14] Naqvi et al found that 41% of patients with malaria associated acute renal failure had Central nervous system symptoms and 81% of patients with Central nervous system symptoms expired as compared to 29 % who survived thereby proving Central nervous system abnormalities to be important predictor of mortality.[15]

Andrea polito et al conducted prospective observational study on septic shock patients and found MRI showed cerebral infarcts in 21 (29%), leukoencephalopathy in 15 (21%), and mixed lesions in 6 (8%). EEG malignant pattern was more frequent in patients with ischemic stroke or leukoencephalopathy.[16] A Study conducted by Kalita et al included 24 patients with dengue virus. EEG revealed theta to delta slowing in 11 out of 20 patients. Five of these patients had seizures, 12 altered sensorium, 3 hypotension, 9 CSF pleocytosis, 3 moderate to severe liver dysfunction and 2 had MRI abnormalities who had Japanese encephalitis virus coinfection.[17]

R. A. Fearnley et al reported a case of Influenza A-induced rhabdomyolysis causing Acute kidney injury in a young adult which was further complicated by posterior reversible encephalopathy syndrome.[18]

A cross sectional study conducted by Misra et al on 37 patients with scrub typhus and neurological changes showed Thirty-one (84%) patients had impaired consciousness, and six were deeply comatose (GCS score ≤8). Eight patients presented with status epilepticus. MRI revealed meningeal enhancement in only 1/25 (4%) patient and EEG showed generalised slowing in 6/28 (21.4%). Among 31 patients with altered sensorium, CSF studies were conducted on 28. Nineteen patients had meningoencephalitis and 9 had encephalopathy.[19]

Sam Alfred et al observed in a study on ethylene glycol poisoning that out of 7 patients with anuric acute renal failure 3 developed significant cranial neuropathies with bulbar palsy in the second week post ingestion. One patient died with cerebral oedema and a progressive encephalopathy. 2 with normal renal function and a moderate metabolic acidemia (ph range 7.2-7.28) remained well without neurological sequale.[20]

HUS is the most common cause of Acute kidney injury in children but is increasing being diagnosed in adults. In a study on pts with HUS Nathanson et all found pts had stuporous coma, protracted severe seizures, and other other neurologic defect including pyramidal syndrome, hemiplegia or hemiparesia, and extrapyramidal syndrome and coma.[21] Steinborn et al studies 57 patients with clinically proven HUS. In six patients, pathological changes within the basal ganglia were found on CT and MRI.[22] Karin Weissenborn found that among 48 patients of HUS All but 1 showed neurologic symptoms. Focal neurologic signs like double vision, difficulties in finding words, or hyperreflexia were present in 23, additional deficits in orientation, attention, memory, or constructive abilities in 9, and marked impairment of consciousness in 15. MRI showed brainstem, midbrain, thalamus, corpus callosum, and white matter lesions in half of the patients, predominantly in diffusion-weighted images. General slowing but no focal alteration was found in half of the patients examined by EEG.[23]

Grażyna Zarzycka-Lindner, et al reported a case of 42-year-old man who developed symptoms of Guillain-Barre syndrome (GBS) 5 days after acute renal failure (ARF) occurred.

The pharmacokinetics of drugs undergo change in renal injury hence neurological side effects of drugs are expected to be precipitated in Acute kidney injury.

D Chatellier et al reported 5 patients suffering from acute renal failure treated with cefepime presenting with seizures and coma.[24]

6.3 AIMS AND OBJECTIVES OF THE STUDY

- Determine what percentage of patients with acute kidney injury develop neurological changes
- Describe The Pattern Of Neurological Manifestation in Acute kidney injury

7.0 MATERIALS AND METHODS

7.1 SOURCE OF DATA

A prospective Observational Study Will Be Conducted at Dayanand Medical College And Hospital, Ludhiana Which Will Include Patients Diagnosed With Acute kidney injury.

- Comprehensive clinical examination will be documented including GCS along with blood counts, chemistries and other relevant investigations on a proforma.
    - Neurological changes will be assessed by baseline scores and neurological examination will be done on day 0 and day 7 of admission.
    - The second follow up will be done earlier if any patient has no neurological features on presentation but develops the same before day 7 of admission.
- To navigate covid precautions any patient included in study who develops covid pneumonitis will be examined by the resident doctor on duty(after being explained and trained on performa by the candidate

7.2 METHOD OF COLLECTION OF DATA

Inclusion Criterion:

1. All Patients >18 Years of either gender admitted in DMCH and diagnosed with Acute kidney injury.

Exclusion Criterion:

- Patients With Underlying Chronic Kidney Disease As Defined By KDIGO Criteria
- Post Renal Transplant
- Patients On Anti Epileptic Maintenance Treatment
- Patients On Mood Stabilizers, Anti Depressants, Antipsychotics
- Survivors Of Brain Tumors
- Patients With History Of Neurosurgical Procedures.

Number of Cases: Total number of patients included in the study will be the first 200 satisfying the inclusion criteria.

Outcome:

The primary Outcome of study will be to see the Spectrum of Neurological Changes in Acute kidney injury.

The secondary outcomes will be to assess any correlation in neurological severity in progressive stages of Acute kidney injury

Statistical Analysis:

Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

- CBC, FBS, Serum Urea, Creatinine, Serum Electrolytes, ABG
- As Per Indications The Following Investigations Will Be Done- CT/MRI /CSF Analysis/EEG /NCV AND EMG.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Yes

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PROFORMA

Name

Age

Sex

Presenting complaints-

ICU Stay-YES/NO

Comorbidities-hypertension

-diabetes mellitus 2

- CAD

Any nephrotoxic drugs prior to admission--YES/NO

Any neurotoxic drugs prior to admission-YES/NO

H/O Alcohol/smoking/substance abuse-YES/NO

Family history of neurological disease-YES/NO

Family history of kidney disease-YES/NO

Mechanical ventilatory support--YES/NO

Ionotropic support —YES/NO

qSOFA

	Day 0	Day 7
qSOFA		

Table 5 Sequential Organ Failure Assessment (Quick) qSOFA

Assessment	qSOFA score
Low blood pressure (SBP ≤100 mmHg)	1
High respiratory rate (≥22 breaths/min)	1
Altered mentation (GCS ≤14)	1

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AKI DIAGNOSIS

Stage	Serum creatinine	Urine output
1	1.5-1.9 times baseline OR ≥0.3 mg/dl (≥26.5 µmol/l) increase	<0.5 ml/kg/h for 6-12 h
2	2.0–2.9 times baseline	<0.5 ml/kg/h for ≥12 h
3	3.0 times baseline OR Increase in serum creatinine to ≥4.0 mg/dl (353.6 µmol/l) OR Initiation of renal replacement therapy OR, in patients <18 years, decrease in eGFR to <35 ml/min per 1.73 m^2	<0.3 ml/kg/h for ≥24 h OR Anuria for ≥12 h

Urine output **Blood urea**

Serum creatinine –

AKI STAGE-

Dialysis session-

	DAY 0	DAY 7
VITALS		
Blood pressure-		
Heart rate		
Respiratory rate		
Saturation oxygen		
	DAY 0	**DAY 7**
Lab values		
TLC		
DLC		
Hb		
Platelet count		
Sodium		
Potassium		
Chloride		
AST		
ALT		
ALP		
Bilirubin		
Total protein		
Albumin		
Calcium		

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Magnesium

Phosphorus

Parathyroid (if done)

Urine R/E

UPCr(IF DONE)

USG abdomen-

ABG

	DAY O	DAY 7
pH		
pco2		
pO2		
Lactate		
Hco3		
So2		

Urine culture

Blood culture

Any other culture

NEUROLOGICAL FEATURES

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GLASGOW COMA SCALE	
Eye-opening (E)	
• Spontaneous	4
• To speech	3
• To pain	2
• Nil	1
Best motor response (M)	
• Obeys	6
• Localises	5
• Withdraws	4
• Abnormal flexion	3
• Extensor response	2
• Nil	1
Verbal response (V)	
• Orientated	5
• Confused conversation	4
• Inappropriate words	3
• Incomprehensible sounds	2
• Nil	1
Coma score = E + M + V	
• Minimum	3
• Maximum	15

	Day 0	Day 7
GCS-		

Scenario 1- pt is sedated

Score	Classification	(RASS)
+4	Combative	Overtly combative or violent; immediate danger to staff
+3	Very agitated	Pulls on or removes tube(s) or catheter(s) or has aggressive behavior toward staff
+2	Agitated	Frequent non-purposeful movement or patient–ventilator dyssynchrony
+1	Restless	Anxious or apprehensive but movements not aggressive or vigorous
0	Alert and calm	Spontaneously pays attention to caregiver
-1	Drowsy	Not fully alert, but has sustained (more than 10 seconds) awakening, with eye contact, to voice
-2	Light sedation	Briefly (less than 10 seconds) awakens with eye contact to voice
-3	Moderate sedation	Any movement (but no eye contact) to voice
-4	Deep sedation	No response to voice, but any movement to physical stimulation
-5	Unarousable	No response to voice or physical stimulation

https://doi.org/10.1371/journal.pone.0207174.t001

	DAY 0	DAY 7
RAAS score		

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PATIENT IS DELIRIOUS

CAM-ICU Worksheet

Feature 1: Acute Onset or Fluctuating Course	Score	Check here if Present
Is the pt different than his/her baseline mental status? OR Has the patient had any fluctuation in mental status in the past 24 hours as evidenced by fluctuation on a sedation scale (i.e., RASS), GCS, or previous delirium assessment?	Either question Yes →	☐
Feature 2: Inattention		
Letters Attention Test (See training manual for alternate pictures) Directions: Say to the patient, "I am going to read you a series of 10 letters. Whenever you hear the letter 'A,' indicate by squeezing my hand." Read letters from the following letter list in a normal tone 3 seconds apart. S A V E A H A A R T Errors are counted when patient fails to squeeze on the letter "A" and when the patient squeezes on any letter other than "A."	Number of Errors > 2 →	☐
Feature 3: Altered Level of Consciousness		
Present if the Actual RASS score is anything other than alert and calm (zero)	RASS anything other than zero →	☐
Feature 4: Disorganized Thinking		
Yes/No Questions (See training manual for alternate set of questions) 1. Will a stone float on water? 2. Are there fish in the sea? 3. Does one pound weigh more than two pounds? 4. Can you use a hammer to pound a nail? Errors are counted when the patient incorrectly answers a question. Command Say to patient: "Hold up this many fingers" (hold 2 fingers in front of patient). "Now do the same thing with the other hand" (do not repeat number of fingers). If patient is unable to move both arms, for second part of command ask patient to "Add one more finger." An error is counted if patient is unable to complete the entire command.	Combined number of errors > 1 →	☐

Overall CAM-ICU Feature 1 plus 2 and either 3 or 4 present = CAM-ICU positive		
	Criteria Met →	☐ CAM-ICU Positive (Delirium Present)
	Criteria Not Met →	☐ CAM-ICU Negative (No Delirium)

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	DAY 0	DAY 7
CAM ICU		

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PT IS CONSCIOUS

Mini-Mental State Examination (MMSE)

Patient's Name: _____ Date: _____

Instructions: ***Ask the questions in the order listed. Score one point for each correct response within each question or activity.***

Maximum Score	Patient's Score	Questions
5		"What is the year? Season? Date? Day of the week? Month?"
5		"Where are we now: State? County? Town/city? Hospital? Floor?"
3		The examiner names three unrelated objects clearly and slowly, then asks the patient to name all three of them. The patient's response is used for scoring. The examiner repeats them until patient learns all of them, if possible. Number of trials: _____
5		"I would like you to count backward from 100 by sevens." (93, 86, 79, 72, 65, ...) Stop after five answers. Alternative: "Spell WORLD backwards." (D-L-R-O-W)
3		"Earlier I told you the names of three things. Can you tell me what those were?"
2		Show the patient two simple objects, such as a wristwatch and a pencil, and ask the patient to name them.
1		"Repeat the phrase: 'No ifs, ands, or buts.'"
3		"Take the paper in your right hand, fold it in half, and put it on the floor." (The examiner gives the patient a piece of blank paper.)
1		"Please read this and do what it says." (Written instruction is "Close your eyes.")
1		"Make up and write a sentence about anything." (This sentence must contain a noun and a verb.)
1		"Please copy this picture." (The examiner gives the patient a blank piece of paper and asks him/her to draw the symbol below. All 10 angles must be present and two must intersect.)
30		TOTAL

(Adapted from Rovner & Folstein, 1987)

Source: www.medicine.uiowa.edu/igec/tools/cognitive/MMSE.pdf

1

Provided by NHCQF, 0106-410

	DAY 0	DAY 7
MMSE		

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ALTERATIONS IN CEREBRAL FUNCTION

		DAY 0	DAY 7
Concentration, fatigue	Serial seven test		
Myoclonus/twitching	Yes /No		
Delusion/hallucination	Yes /No		
Posture			
Asterixis	Yes /No		
Seizure	Yes /No If yes -Type		
Sleep inversion	Yes /No		
aphasia	Yes /No		

CRANIAL NERVES INVOLVEMENT

		Day 0	Day 7
CN1	Sense of smell		
CN2	Visual field-quadrant method Vision Pupils		
CN3,4,6	Nystagmus Extraocular movements		
CN5	Chew/bite/lateral jaw movements Sensory function		
CN 7	Motor'-smile/frown /raise eyebrows		
CN8	Rinne test Weber test		
CN 9,10	Gag reflex Swallowing Uvula movement		
CN 11	Turning head Upward shoulder shrug		
CN 12	Tongue movement		

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MUSCULAR DISORDERS

		Day 0	Day 7
Power	GRADING ICMR Upper limb R/L Lower limb R/L Trunk		
Tremor/involuntary movements	Yes/No		
Wasting	Yes/No		
Fasciculation	Yes/No		

SENSORY SYSTEM

	DAY 0	DAY 7
Touch, pin prick sensation,vibration		
vibration		
Stereognosis		
2 point discrimination		

DEEP TENDON REFLEX

Reflexes	Day 0	Day 7
Jaw jerk		
Triceps		
Supinator		
Knee		
Ankle		
Abdominal		
Plantar		

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Postural fainting-YES/NO		
Loss of urinary control -YES/NO		
Night diarrhea-YES/NO		

	Day 0	Day 7
Neck rigidity-yes/no		
Gait abnormality yes/no		

ADDITIONAL INVESTIGATIONS AS AND WHEN INDICATED

CT HEAD	
MRI	
EEG	
NCV/EMG	

CSF ANALYSIS- as and when required

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Signature of Supervisor

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o.....
.....am willing to participate in the study. I am ready to participate in this study on my own **"SPECTRUM OF NEUROLOGICAL MANIFESTATIONS IN ACUTE KIDNEY INJURY: A PROSPECTIVE OBSERVATIONAL STUDY IN TERTIARY CARE CENTRE"** I affirm that I have not been given any financial incentive and no pressure has been put on me to give the consent. The purpose of this research has been explained to me and I believe that it is for the benefit of science and mankind. I have been explained about the risk in this research and I understand that the risk is very minimal. I have been explained the method of participating in this research work.

I can choose to opt out of this research at any time and my opting out will not have any effect on my treatment.

....
Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"SPECTRUM OF NEUROLOGICAL MANIFESTATIONS IN ACUTE KIDNEY INJURY: A PROSPECTIVE OBSERVATIONAL STUDY IN TERTIARY CARE CENTRE"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"SPECTRUM OF NEUROLOGICAL MANIFESTATIONS IN ACUTE KIDNEY INJURY: A PROSPECTIVE OBSERVATIONAL STUDY IN TERTIARY CARE CENTRE" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

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ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

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ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

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ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ